TRANSACTIONS

AMERICAN ACADEMY
of
OPHTHALMOLOGY
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SEPTEMBER - OCTOBER
1957

Volume 61 No. 5

ANNUAL MEETING, OCTOBER 13-18, 1957, CHICAGO

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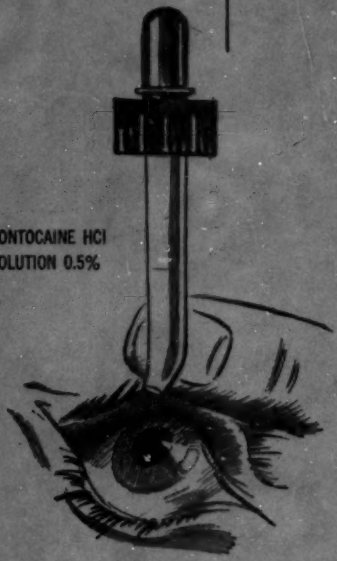

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TRANSACTIONS

American Academy of Ophthalmology and Otolaryngology

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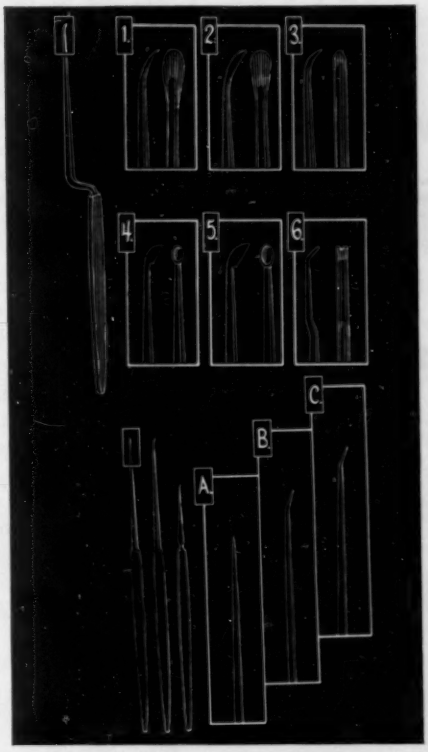

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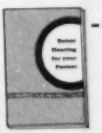

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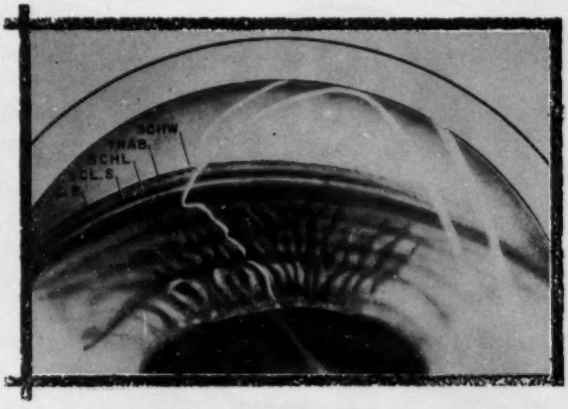

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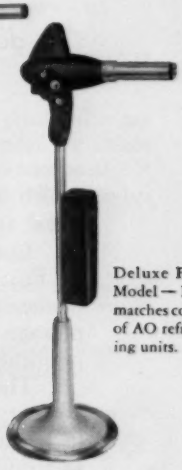

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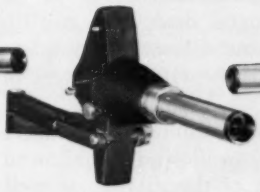
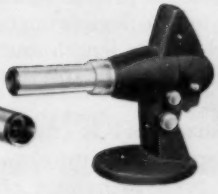


Unit Model—Mounts by bracket on refracting unit.



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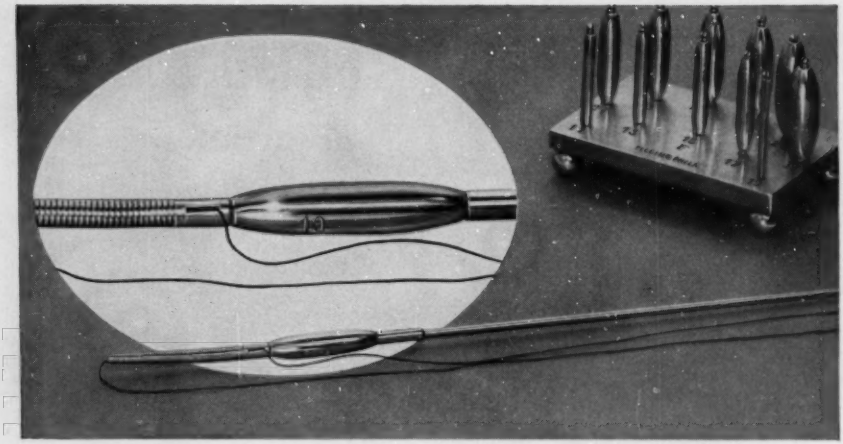

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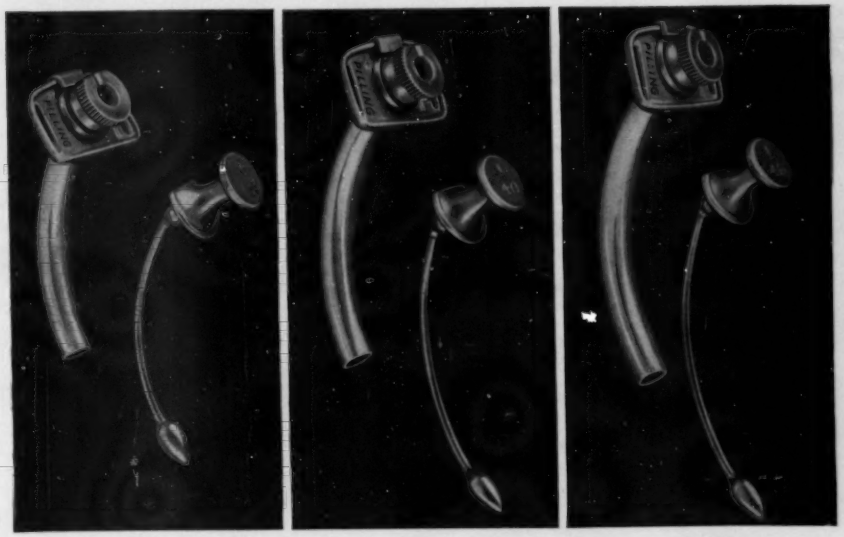

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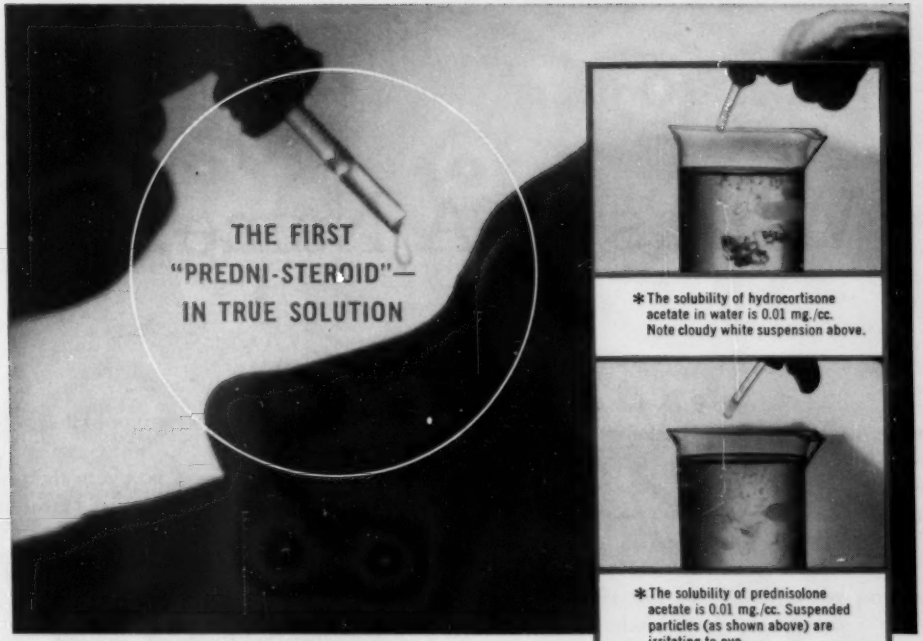

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We need a word or term which not only describes what bifocals do but, *at the same time*,

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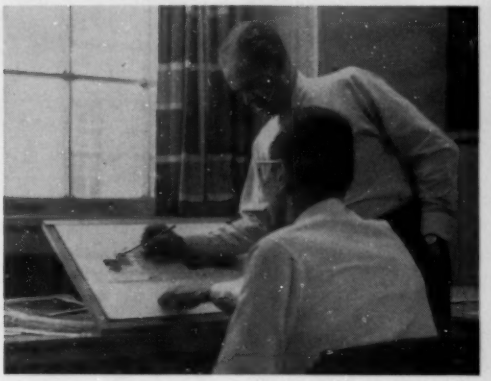

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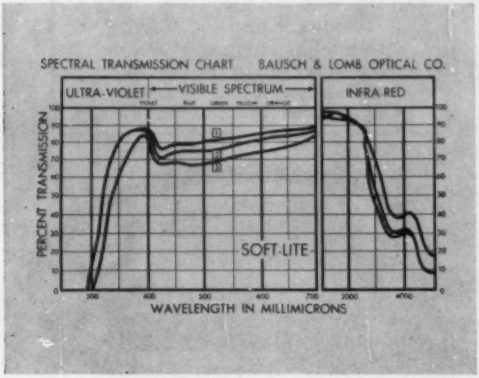

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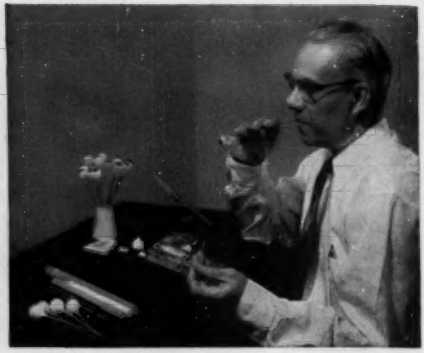

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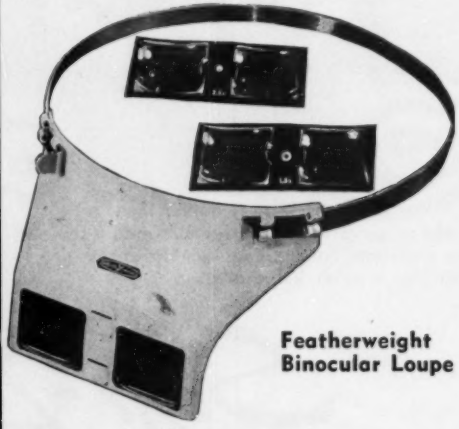

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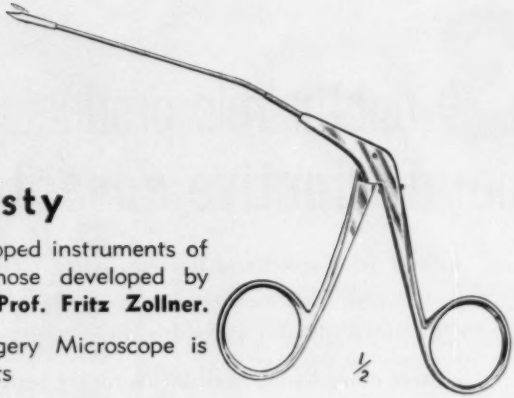

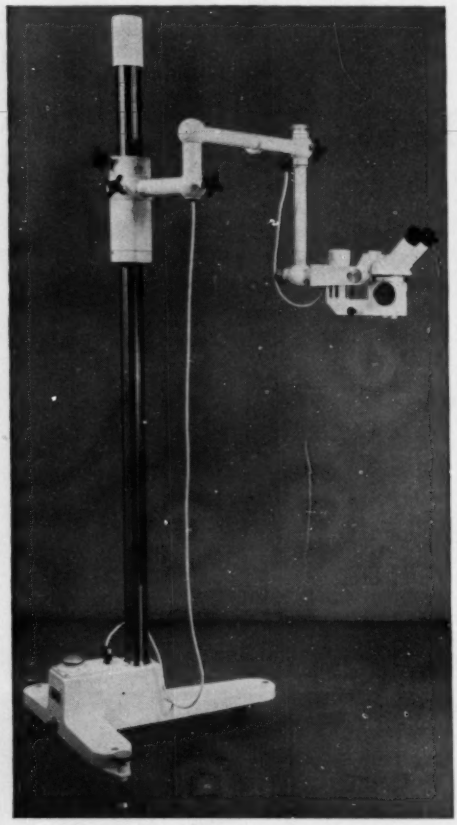

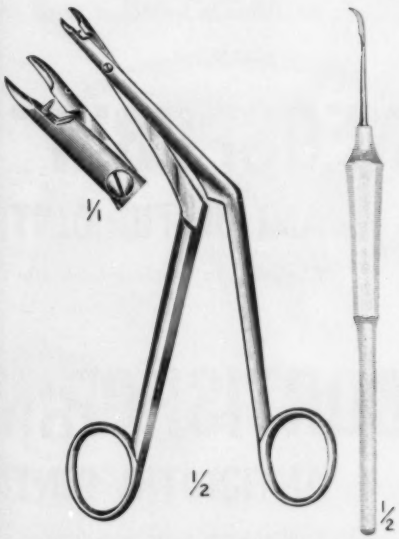

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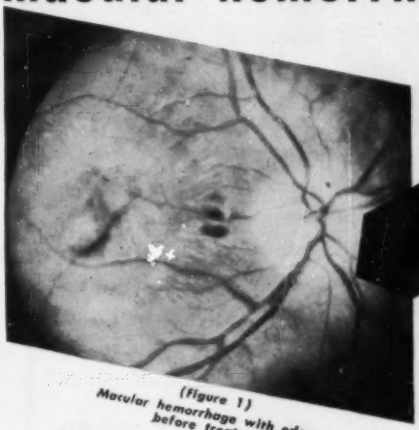

(Figure 1)
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(Figure 2)
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1. *Am. J. Ophth.* 42:771, 1956.
2. *Am. J. Digest Dis.* 22:5, 1955.
3. *Med. Times* 84:741, 1956.
4. *Cecil's Textbook of Medicine*, 7th ed., 1947, p. 1598.

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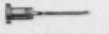

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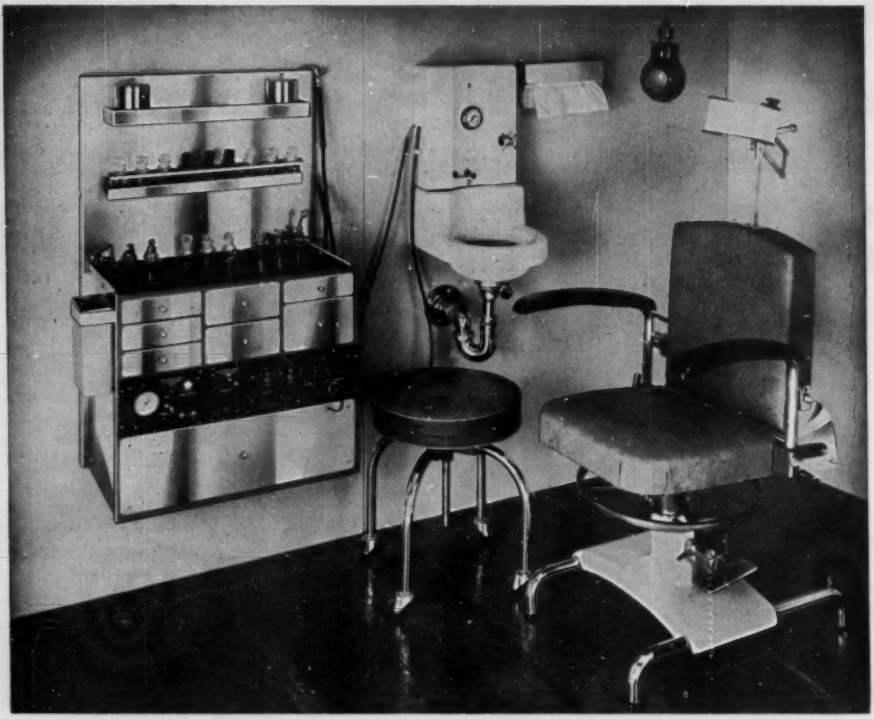

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1. Rasgorshek, R. H. and McIntire, W. C.: Am. J. Ophth. 40:34 (July) 1955. • 2. Ehrlich, L. H.: N. Y. State J. Med. 53:3015 (Dec. 15) 1953. • 3. Gettes, B. C.: A.M.A. Arch. Ophth. 51:467 (April) 1954. 4. Council on Pharmacy and Chemistry: J.A.M.A. 158:1523 (Aug. 27) 1955. • 5. Stolzer, I. H.: Am. J. Ophth. 36:110 (Jan.) 1953.

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THE NEWER ANTIBIOTICS

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PERHAPS I should apologize, as an internist, for being here. However, it is apparent that the areas treated by ophthalmologists and otolaryngologists serve as major portals of entry for a great many infections; therefore, it was felt by the Program Committee that you might be interested in some of the newer antibiotics as they are being developed.

There seems to be no end to antibiotics as far as the ability to find them is concerned; that is, to find microbes producing new and unusual chemicals with antimicrobial activity. However, the road to the development of clinically useful antibiotics is, indeed, a very rocky one. Since most of the infections that we see at the present time are reasonably well controlled, one sometimes wonders why there is still such an avid search for new antibiotics. The main reason for the continuing search is that there are a large number of rather *incurable* infections, particularly chronic infections, and of infections due to resistant organisms. The control of one type of infection in particular, staphylococcic infection, has become a problem of increasing importance.

I shall review several antibiotics which have reached the clinical experimental level and have either reached, or may soon reach, the commercial market; then I shall try to show how they may help to solve the problems that exist in the treatment of infection with antibiotics.

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Presented at the Sixty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 14-19, 1956, Chicago.

NOVOBIOCIN

You probably know novobiocin by one of its trade names, Albamycin or Cathomycin. Organisms which are extremely sensitive to less than a microgram per milliliter of novobiocin include the staphylococci, Corynebacterium, Hemophilus pertussis, and Hemophilus influenzae. The pneumococci and streptococci of the *alpha* type are slightly less sensitive. They are occasionally sensitive to less than one microgram, and usually sensitive to less than 10 micrograms per milliliter of the antibiotic. Proteus vulgaris and some types of Pasteurella are sensitive to a minimal inhibitory concentration of more than one but less than 10 micrograms per milliliter of novobiocin. Proteus vulgaris is a common cause of cystitis and is a major factor in several chronic respiratory diseases. Its sensitivity to novobiocin was one of the main reasons that further study was indicated. Other organisms, including Streptococcus faecalis, Klebsiella, and Brucella, are out of the range in which one can hope to get a beneficial effect.

Animal infections by staphylococci (group A), streptococci, pneumococci, Pasteurella multocida, Proteus vulgaris, and Hemophilus pertussis have been treated successfully with novobiocin, but such treatment has failed in the gram-negative rod infections, such as those by Salmonella, Shigella, Klebsiella, and Escherichia coli.

In the clinical situation one of the marked potentials of worth of this drug has been its excellent absorption when given orally (table I). A dose as small as 5 milligrams per kilogram will pro-

duce serum levels as high as those customarily seen with many of the commonly used antibiotics. With a high dose, 25 milligrams per kilogram, which is not a great dose of an antibiotic, one sees levels as high as 30 to 40 micrograms per milliliter. Even at the end of eight hours there is still appreciable activity. Theoretically, this looks like a very promising drug; it has had clinical trials in pneumococcal, staphylococcal, and streptococcal infections.

In streptococcal infections—you may remember that streptococci have intermediate sensitivity—novobiocin has not worked as well as penicillin and some of the other established antibiotics.

TABLE I

NOVOBIOCIN

SERUM CONCENTRATION IN MCG/ML	ORAL DOSE (SINGLE)		
	5 MG/KILO.	10 MG/KILO.	20 MG/KILO.
At 1 hour	5.6	5.6	33.0
At 8 hours	7.3	15.3	14.4

Route: Oral; Intramuscularly or intravenously
Clinical use: Pneumococcal infections
Staphylococcal infections
Streptococcal infections

Clinical results have been somewhat discouraging, considering the apparent efficacy of novobiocin in animals, and the fact that it looked so promising from a pharmacologic point of view. Pneumococcal infections have not responded as consistently to novobiocin as they do to penicillin. They respond more as they do to the sulfonamides, with persistence of the carrier state in the upper respiratory tract and somewhat more frequent relapses. Staphylococcal infections have responded well. Some very serious staphylococcal infections, resistant to all other types of antibiotics, have responded. However, there has been a major problem. The staphylococcus has shown its ability to develop resistance to this drug while it is being used. In a relatively short period of time—five to ten days—people with staphylococcal abscesses and septicemias have developed resistance to the drug while it was being administered. The matter of resistance will be discussed later, when we talk about the problems of using drugs in combinations. Such resistance has been a limitation even in this one area in which the drug shows most promise.

However, it is effective. It is probably somewhat more effective than the sulfonamides, but it is not considered the drug of choice.

The major problems encountered in treatment with novobiocin has been its marked propensity for producing rashes. The rash induced by novobiocin looks almost exactly like that due to a sulfonamide. Some patients become sensitized and respond immediately to a second course of the drug, which is very similar to the way sensitive patients react to the sulfonamides. The reports on the incidence of rash vary. It has been reported to be as high as 18 per cent in a given group of patients, but most of the figures cited in the literature are somewhere between 3 and 10 per cent. Whether the occurrence of rash is contingent on some impurity in the compound or whether the reaction is to the

drug itself is a question which has not been completely answered. Attempts are being made to modify the drug structure and to increase its purity so that the incidence of reaction can be reduced.

Leukopenia has occurred, as one might expect when a drug gives this type of toxic reaction, but reported cases have been rare. There is no way to estimate their frequency, but it is probably less than, or at least of the same order of magnitude as the frequency of leukopenia due to the sulfonamides.

It has been shown that the staphylococcus becomes resistant to novobiocin under treatment. There are also a fair number of resistant strains of staphylococci already present in the environment —about 5 per cent. In one hospital population we were able to document the accumulation of resistant strains favored by the extensive use of the drug, which was similar to the spread, in the hospital environment, of resistance of staphylococci to other drugs. Problems of both types have been met in the resistance of staphylococci to novobiocin, namely, development of a resistant strain in a patient receiving the drug, and accumulation of prior resistant strains in the environment.

One other factor which may account for our difficulty with novobiocin is that there are inhibitors in the serum. Unless one gets a fairly high concentration, 8 micrograms per milliliter or more, the inhibitors of concentrated serum can neutralize the effect. This may explain why the results have not substantiated the pharmacologic predictions.

SPIRAMYCIN

Spiramycin is the discovery of a French investigator; it has been used extensively in European clinics. It has not been used as extensively in America because it is very similar to erythromycin. However, it does not completely cross-react with erythromycin. In general, organisms routinely isolated as resistant to erythromycin are sensitive to the action of spiramycin. However, there is enough cross-resistance so that if one uses spiramycin and produces an accumulation of resistant strains in the hospital, one generally gets an increase in the erythromycin-resistant population concurrently.

Organisms sensitive to spiramycin include those pathogens commonly found in the upper respiratory tract: streptococci (group A), pneumococci, Neisseria and Corynebacterium. A little less sensitive to the drug, but still on a treatable level, are staphylococci and enterococci, the streptococcus originating in the gastrointestinal tract. Klebsiella and Salmonella are not sufficiently sensitive to the drug for it to be of practical usefulness in treating infections due to them.

Animals treated with spiramycin for streptococcal, staphylococcal, pneumococcal, rickettsial, and treponemal infections have shown good response.

Spiramycin is absorbed rather poorly; the dose should be as high as 100 milligrams per kilogram daily to get adequate levels. Even these levels are somewhat erratic, as the range from 0.06 to 8.0 indicates. Spiramycin can only be given orally. There is no parenteral form developed at the present time.

Spiramycin has been shown to be efficacious clinically in streptococcal infections, including scarlet fever and otitis media. It is probably not as effective as penicillin, although it is difficult to make a controlled study to prove this point at the present time. However, the incidence of positive carriers at the end of a seven-day course of treatment is higher for this drug than it is for penicillin.

The use of spiramycin in the treatment of pneumococcal infections, par-

ticularly pneumonia, has been studied, and these infections have responded about as well to spiramycin as to any other antibiotic.

Even though the offending organism may be erythromycin-resistant, staphylococcal infections have been treated successfully with this drug. This is probably a major reason for the introduction of spiramycin into the clinical field at the present time.

Neisserian infections have been treated successfully with spiramycin.

Toxicity has been low. One seventh of the patients treated with spiramycin complain of some looseness of stools. However, there have been no serious diarrheas, and no allergic reactions have been reported to the present time.

The resistance problem has been the same as it has been for almost every drug introduced. Staphylococci have become resistant under treatment. The resistant staphylococci in the environment have proliferated and have spread throughout the hospital environment.

OLEANDOMYCIN

Oleandomycin, known previously as Amimycin® and PA 105®, is related to erythromycin and is similar in its activity. This drug has a fairly wide range of activity against staphylococci, group A streptococci, and Listeria. The sensitivity of Listeria to the other drugs mentioned has not been tested, so it is not clear whether the sensitivity of oleandomycin represents a broadening of the spectrum. Potentially, oleandomycin should be effective in the treatment of any infection by staphylococci, streptococci (group A), Erysipelothrix, Corynebacterium, Listeria, or Hemophilus influenzae. These organisms are sensitive to a minimal inhibitory concentration of less than one microgram per milliliter. Pneumococcus, Streptococcus faecalis, Neisseria, Clostridia, and Brucella are sensitive to more than one but less than 10 micrograms per milliliter. Studies of effect of oleandomycin on animal infections are limited. Streptococcal and staphylococcal infections have responded. This drug has been used in doses of about 40 milligrams per kilogram. The drug is now available only for administration by the oral route.

Clinically, oleandomycin has been tried in infections by pneumococci, Hemophilus influenzae, and staphylococci, all of which have responded well. The effect of the drug on staphylococcal infections has not been studied as extensively as the effects of other drugs, and the propensity of this drug to produce resistant strains has not been as well documented. It is true that this drug will cross with erythromycin to a certain extent, and it is likely that the staphylococcal resistance problem will develop as the drug is better evaluated.

Toxicity has been reported as low. Some minor gastrointestinal disturbances have been reported.

This drug has been used most frequently in combination with another drug, and probably it will be marketed in one of these combinations. A combination with one of the tetracyclines (Terramycin) has shown promise of some beneficial effects in the laboratory, and it is felt that this combination may work better against staphylococci than either drug used alone. This remains to be proved. However, because both drugs are manufactured by the same company, a combination of the two is probable.

Again I should like to emphasize that oleandomycin is similar to erythromycin in its range of activity, but it has unique pharmacologic properties which make it appear to be a useful drug.

SYNAMATIN

Synamatin is a penicillin, interestingly enough. The chemical structure of this and other penicillins do not differ greatly; ring structure is identical and the variations occur in the sidechains. However, there is considerably more difference in the action of this penicillin and other penicillins that have been studied, such as penicillin O, penicillin G, and penicillin X. This particular agent has a marked accentuation of the anti-gram-negative property of penicillin, being most effective against Salmonella and some strains of Proteus, particularly Proteus mirabilis. However, it is also active against most cocci. At present the only form available is for parenteral administration. It is generally recommended that a dose of 250,000 units be given every three hours intramuscularly. The volume of the dose as currently given is 4 cc. This drug still has a relatively limited usefulness because the method of administration has not been developed.

In Salmonella infections, this may be the drug of choice at the present time, and it is included in this discussion because it has proved so effective in the treatment of such infections. Moreover, Proteus mirabilis is very sensitive to oleandomycin, and you may find this drug very efficacious in the chronic infections by this organism which may be plaguing some of your patients. Proteus strains really divide in their reaction to drugs; mirabilis infections are generally sensitive to drugs in the penicillin family, and the vulgaris infections are more sensitive to those in the tetracycline family.

VANCOMYCIN

Vancomycin is a drug with a fairly limited range of activity. It has been introduced mainly because of its antistaphylococcal properties. It can be given only parenterally, either intravenously or intramuscularly. The recommended dose has been 500 milligrams twice daily, and the results of its use have been relatively good. However, its spectrum is similar to those of other well-established antibiotics, and its chief promise lies in the field of staphylococcal infections.

ANTIFUNGAL AGENTS

I have included two antifungal agents because fungus infections occasionally occur in the respiratory tract. Unfortunately, there is no really good antifungal agent available for most of these infections. Nystatin, which is marketed under the trade name of Mycostatin and, in combination with tetracycline, as Mysteclin, can only be given orally. This limitation is due to the fact that its absorption is poor and its antifugal action is entirely local. It is employed to suppress fungi in the gastrointestinal tract and thereby reduce the complications due to overgrowth of monilial organisms nonsusceptible to other drugs, but it is a good agent only for local medication.

The toxicity, however, because of the lack of absorption, is quite low.

Amphoteracin A and B are agents which at present can only be given orally. However, they may be absorbed a little better than nystatin and therefore offer some promise. In mice, amphoteracin B is more active against certain fungal infections than is nystatin, and it appears to be a very promising antifungal agent.

Two big problems exist in the development of antibiotics—the problems of resistance and of chronic infections. Are these new drugs the answer to these problems?

Problems of resistance are of two types. Whenever an agent is introduced, there are usually mutants in the community which are already resistant to the agent. These may spread under a locally favorable condition in a hospital situa-

tion so that a high incidence of resistant strains develops among the carriers and a high incidence of acquired resistant infections occur in the hospital. The development of resistant strains of staphylococci has been reported often, and it is quite probable that this development of resistant strains is also true of most of the gram-negative rods. We have been interested in this area as far as post-tracheotomy infections in poliomyelitis patients are concerned, where there is a high incidence of resistant infection by the staphylococci and occasionally by the gram-negative rods. These agents are resistant to most of the antibiotics that have been used extensively in a particular hospital.

In addition, there is the problem of a new strain becoming resistant while the patient is receiving the drug. This is a much less common phenomenon. It occurs primarily with staphylococci and tubercle bacilli. It occurs more with some antibiotics than with others. As we have indicated, it has been reported to have occurred already with novobiocin and spiramycin, and it probably will occur with oleandomycin.

The second problem is what we classify as untreatable infections. There are chronic infections deep in the tissues, in which the organisms may not be highly resistant but are resistant enough that one cannot eradicate them from the tissues when one must also consider the problem of penetration and the fact that the drugs are mainly bacteriostatic. New agents are being sought in order to try to get more effective concentrations in the tissues. The need for more bactericidal action has been one of the big stimulants in the development of new antibiotics.

The resolution of these two problems depends upon these four possibilities. One is the use of combinations. It is possible that certain combinations delay development of resistant mutants in clinical situations. As we indicated, if novobiocin is used in combination with a second agent to which the staphylococci are sensitive, such as chloramphenicol, the development of resistance to novobiocin may be delayed and the effectiveness of the drug extended. This type of benefit to be derived by a combination of drugs is not yet documented except in the case of tuberculosis.

We have recently made a study of the use of novobiocin and spiramycin together in treating almost all of the patients in a single hospital population. We were able to show that the development of the dissemination of resistant strains was delayed for approximately two months. At the end of that time, however, the resistant mutants broke through and finally there was, by the end of the six months, about 20 to 30 per cent resistance to both spiramycin and novobiocin.

For some time the postulation has been that if we could use two new drugs in combination from the beginning, we could greatly reduce the incidence of resistant forms as they spread through the community. This postulation was not borne out by the study. All we can say is that we delayed resistance briefly. Probably nothing is accomplished by such a short delay, since we ended up with strains resistant to both agents. The two agents could have been used equally well in sequence.

Frequent change of antibiotics certainly will help to answer the resistance problem. The frequent change that has been instituted in some hospitals has allowed some agents to regain their original sensitive properties; one of the most noteworthy being chloramphenicol. This drug was replaced by other agents fairly early because of its toxicity. The resistance of staphylococci that were disseminated in certain clinics has de-

creased markedly, so that there is considerably less resistance to chloramphenicol now than there was in 1952.

One reason new antibiotics are being introduced is that the new antibiotic takes the pressure off the old ones, so to speak, and allows the resistant strains to equilibrate at a lower level. Thus, when agents are used infrequently, it is less likely that organisms resistant to all the antibiotics at the same time will accumulate. That is the third point.

It is probable that we will not see again the development of saturated populations in hospitals, such as we saw with cycline and penicillin, simply because the number of agents now available and being used in a random fashion in hospital populations does not allow sufficient concentration of patients receiving any one agent to build up these enormously high carrier rates. It is likely, just as with erythromycin, that resistant forms will stabilize pretty much at a 10 per cent level in most hospitals.

As we produce more of these agents, probably we shall see some organisms resistant to some specific agents but very few organisms resistant to all of the agents, and a lower incidence of organisms resistant to any one agent in the population.

LOCAL THERAPY

The last point that I shall discuss is local therapy. Local therapy is a matter of great concern to the ophthalmologist and the otolaryngologist. Local therapy, as I see it, has one major problem—that is to get the antibiotic in contact at the site of the organism for a sufficient period to do the job. This depends primarily on the rapidity of the action and the ease of administering it directly to the diseased tissues. When it can be kept in contact with the organism at the site of the infection, it works quite well; consequently, ointments, which allow gradual penetration of the antibiotic, are better vehicles for therapy than troches, which lose their antibiotic action almost immediately. Unfortunately, the rapidity of the action is all important. The agents we have talked about are all primarily bacteriostatic. They require long contact to suppress the infection; long enough for the host to do something about it. Consequently, they are not very good agents for local therapy. The best agents for local therapy are those which are bactericidal and can reduce the colony count appreciably in the first half hour or hour of exposure to the organisms.

Unfortunately, a great many new antibiotics are limited in their usefulness because they can be used only locally. This plays a part of the economics of the development of antibiotics; an antibiotic has to be pretty much all-purpose, and its oral administration in the treatment of most of the common infections should be practicable if it is to make a place for itself. We do hope that there will be more and more use of some of the agents which are not suitable for parenteral use in local infections. Perhaps the most encouraging local use of antibiotics has been that reported by the Australian workers who were able to reduce the incidence of resistant staphylococci carriers in a hospital, by the local use of bacitracin and neomycin ointment in the nose. This type of study will have to be repeated and confirmed. However, it is the first indication that something is being done to solve the problems of the spread of resistant organisms.

APHAGIA FROM POLIOMYELITIS: TREATMENT BY DENERVATION OF THE CRICOPHARYNGEUS MUSCLE

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THE purpose of this paper is to present a relatively new surgical technique for the relief of persistent aphagia resulting from acute poliomyelitis. Complete inability to swallow is a rare complication of poliomyelitis. Since 1950, I have observed only two such cases in 4975 admissions at the Elizabeth Kenny Institute in Minneapolis. Inability to swallow is so distressing to the patient that I feel justified in describing a simple surgical procedure which, if performed, will again allow the patient to swallow. During the past year, I have discussed this problem at several meetings of otolaryngological societies and have learned that the large poliomyelitis centers have at least one or two cases of aphagia included in their rehabilitation program. Perhaps there are some among you who likewise are faced with this perplexing problem; I would strongly suggest that you employ this simple surgical procedure of cricopharyngeal denervation for the relief of aphagia.

Before presenting the case reports and describing the technique of the operation, let us briefly consider the anatomy, neurology, physiology, and pathology of the cricopharyngeus muscle. The cricopharyngeus muscle was first described as far back as 1717 by Valsalva, a man whose name is most familiar in our specialty. In his original description, Valsalva referred to the cricopharyngeus muscle as a definite anatomic entity, having very distinct features, just as any other striated muscle in the human body. Since his original description, however, succeeding anatomists have not universally recognized this anatomical concept. They have been content to describe the cricopharyngeus muscle as either the lower part of the inferior constrictor muscle of the pharynx, or the upper part of the circular fibers of the esophagus. In a recent classical article describing the anatomy of the cricopharyngeus muscle, which he observed following a special cadaveric preparation technique, Batson[1] confirmed Valsalva's original concept of the cricopharyngeus muscle as a definite anatomic entity. Batson describes the cricopharyngeus muscle as a circular band of horizontal fibers, measuring approximately 20 millimeters in height, which is attached to the cricoid cartilage laterally at the junction of the annular and signet portions of the cartilage. The muscle forms a sling which encloses the mucous membrane of the lowermost portion of the hypopharynx.

The physiologists have demonstrated that the short horizontal fibers of the cricopharyngeus muscle have the ability to contract to one half of their length, which accounts for the tremendous sphincter action of the muscle. During esophagoscopy, this sphincter action is readily observed; Jackson[2] has referred to this area as the cricopharyngeal pinchcock. This state of tonic contracture is the normal quiescent stage of the cricopharyngeus muscle. Negus[4] has demonstrated that the purpose of this contraction is to prevent air from being inspired into the esophagus, where it would serve

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no useful purpose and would only interfere with respiration by decreasing the effective tidal volume. Relaxation of the cricopharyngeus muscle occurs during deglutition, when the bolus of food stimulates the sensory fibers of the vagus as the bolus passes the pharyngeal constrictors. As the cricopharyngeus muscle relaxes, the larynx is closed and respiration temporarily ceases. During this relaxation phase the bolus of food passes from the cricopharyngeus area into the esophagus, whereupon the cricopharyngeus muscle again constricts before respiration recommences. The tonicity of the muscle apparently varies from individual to individual. Many roentgenologists feel that the relaxed state of the cricopharyngeus muscle is an expression of high intrapharyngeal pressure, and that the contracted state is an expression of low pharyngeal pressure.[7]

The innervation of the cricopharyngeus muscle has been quite extensively studied by British investigators. It has been established that the principal nerve supply to the cricopharyngeus muscle arises from the glossopharyngeal nerve and the inferior laryngeal nerve, a branch of the vagus. The pharyngeal plexus, supplying the cricopharyngeus muscle, in addition to the parasympathetic component has a rich supply of fibers arising from the superior cervical ganglion, which form the sympathetic component.[5]

Very little experimental work on the cricopharyngeus muscle has been done using laboratory animals. It has been demonstrated in the cat that a bilateral vagotomy performed high up in the neck, under the tympanic bulla, results not only in a midline paralysis of the vocal cords but also in a complete bilateral pharyngeal paralysis associated with a strong contracture of the cricopharyngeus muscle and cardiospasm.[6] This spasm at both ends of the esophagus, it is claimed, is due to a strong sympatheticotonic contracture at both the cricopharyngeus and cardiac levels of the esophagus. With interruption of the vagus nerve, the parasympathetic component to the cricopharyngeus muscle is destroyed; the sympathetic components arising from the superior cervical ganglion act independently and account for the marked sphincteric action producing aphagia.

Clinically, I have observed several cases of aphagia as a complication of bulbar poliomyelitis. All but two of the cases were temporary; however, those two cases of aphagia persisted more than three years and were not relieved until denervation of the cricopharyngeus muscle was effected. To my knowledge, there has been only one other case of this nature reported in the literature.[3]

Case 1.

The first case concerns a senior student nurse, 22 years old, who was severely stricken with bulbar poliomyelitis during the 1952 epidemic in northwestern Iowa. Because of her inability to cope with her oral-pharyngeal secretions, a tracheotomy was performed three days following admission. Nournishment of the patient was maintained by intravenous feedings during the two-week critical period of her disease. Following the acute phase of the disease, she was fed by means of a gastric gavage tube introduced through the mouth.

Several months after the onset of her disease, the patient was transferred to the Elizabeth Kenny Institute in Minneapolis. Upon admission there, she was extremely emaciated. She was wearing a tracheotomy tube and had to be in the artificial respirator continually. She drooled constantly from the corners of her mouth because of her inability to swallow. She had to be tube fed four times daily. Neurological examination revealed that in addition to the inability to swallow there was paralysis of the right seventh, eleventh, and twelfth cranial nerves.

The patient's general condition improved very slowly because of frequent tracheobronchial complications. It was several months after her transfer to the Kenny Institute before we felt that it was safe to study her swallowing mechanism with a contrast media under fluoroscopy. At that time she was still

being tube fed, and actual measurements of secretions drooling from her mouth would frequently total over a liter during a 24-hour period. Thus, it can be appreciated how miserable this patient was, not only because she was unable to swallow food but also because she was entirely unable to cope with her salivary secretions.

The pharyngogram demonstrated difficulty in initiating the act of swallowing. There was marked limitation in tongue movement and inability of the tongue to propel the contrast media into the pharynx. After the contrast media reached the pharynx, it stagnated in the valleculae and had not passed the cricopharyngeus area one-half hour after ingestion.

A prolonged period of psychotherapy and the employment of various antispasmotic drugs were to no avail. Frequent dilatations of the cricopharyngeus area with the hydrostatic dilator were of no help, and the problem of aphagia persisted. I suggested to this nurse patient that we section her cricopharyngeus muscle, after I learned that Dr. Samuel Kaplan of Beverly Hills, California, had successfully treated a similar case in such a surgical manner. The cricopharyngeus muscle was exposed and sectioned, and within a few days the patient was able to cope with her salivary secretions and to take liquids by the oral route, after being unable to swallow anything for forty months. Gradually she began to swallow solids, and at the present time she can maintain her nutrition by the natural route.

Case 2.

The second case of aphagia complicating bulbar poliomyelitis treated by sectioning the cricopharyngeus muscle was even more dramatic in its response than the first case. Within two weeks after the operation the patient was able to swallow all solids as well as liquids. This was the case of a 10-year-old boy who had likewise developed poliomyelitis during the midwestern epidemic in 1952. The course of the disease in this case was much the same as that in the previous case except that this patient was discharged to his home from the hospital one year following admission. The tracheotomy tube had been successfully decannulated. His nutrition was maintained for an additional two years by means of four daily tube feedings of a high-calory protein formula supplemented with vitamins and minerals. He was subsequently readmitted to the hospital, where the cricopharyngeus muscle was sectioned almost three years after the onset of acute poliomyelitis. Within twenty-four hours the patient was able to swallow and cope with his salivary secretions, which he had been unable to do prior to the operation. On the fourth postoperative day he was able to swallow his entire liquid formula, and by the fourteenth postoperative day he was able to take solids quite well. At the present time he has no difficulty whatsoever with his swallowing mechanisms. He was more promptly rehabilitated than the first patient, I believe, because the muscles which allowed him to initiate the act of swallowing functioned normally. He could normally pass a bolus of food into the pharynx, where it would gravitate to the cricopharyngeus muscle, as shown by the presurgical roentgenogram studies. After the cricopharyngeus muscle had been sectioned, the food then readily passed the relaxed denervated area into the esophagus, as shown by the postsurgical roentgenograms.

The surgical technique of sectioning the cricopharyngeus muscle is simple. The procedure may be performed under either local or general anesthesia. An incision is made along the anterior border of the sternocleidomastoid muscle, extending from the upper border of the thyroid cartilage to the lower border of the cricoid cartilage. The soft tissues are bluntly separated until the posterior borders of the thyroid and cricoid cartilages are exposed and rotated freely outward and laterally. This brings the inferior constrictor and the cricopharyngeus muscles into good view. With an esophagoscope in place, the cricopharyngeus muscle is safely peeled off the pharyngeal mucosa and a section of the muscle removed from the posterior midportion. The surgical wound is then closed in layers, without a drain. Attempts at swallowing may be made after a twenty-four-hour postoperative period.

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DISCUSSION

KURT TSCHIASSNY, M.D., Cincinnati: Dr. Bofenkamp is to be commended for his idea and perfect technique of performing a procedure which is certainly to be regarded as a new contribution.

I want to ask him a question. He mentioned that the cricopharyngeus muscle had the same function as a sphincter muscle. As all sphincters do, this muscle must prevent undesirable objects from entering into a structure which has to be protected. Other sphincter muscles are no different from this one. Whether it is the sphincter muscle of the bladder, the anus, or of the esophagus, they are closed when at rest and they are open only when there is a need for them to be open. If the cricopharyngeal sphincter was not resting during inspiration, air would enter into the digastric tract and the result would be uncontrolled belching.

Alvarez states (J.A.M.A., 120:21-24 [Sept. 5] 1942) that in some psychoneurotic people, the cricopharyngeus muscle is paralyzed. These people inspire air into the esophagus, and belching results. Not only that, but also they have inconvenient flatulence because only part of the air, the oxygen, is absorbed by the stomach and by the intestinal tract; the rest, the nitrogen, goes the road of all meat.

I want to ask Dr. Bofenkamp whether he has observed something like uncontrolled belching in his patients.

DR. BOFENKAMP: Thank you for your kind comments.

I must admit that it was with some fear that I operated upon the cricopharyngeus muscle, knowing that I would violate the structure and function of the muscle.

One of the two operations reported was done a year ago and the other, six months ago. Another patient was operated upon last week. None of these patients has made any complaint relative to flatulence.

CHRONIC FRONTAL SINUSITIS: END RESULTS OF SURGICAL TREATMENT

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BEFORE giving the end results in the surgical treatment of chronic frontal sinusitis using the mucoperiosteal membrane, I believe it wise to review the technique of the procedure for the benefit of those who may not know of the operation.

I believe we all are agreed that the greatest obstacle to the cure of chronic frontal sinus disease is the problem of handling the nasofrontal duct. In my opinion, efforts to preserve the nasofrontal duct are the chief cause of failure in operations on the frontal sinus.

THE OPERATION

I prefer local anesthesia.

Sewall[3,4] advocates the complete removal of the nasofrontal duct. He makes an opening and covers it with a mucoperiosteal membrane from beneath the nasal process of the maxillary bone and a portion of the nasal bone.

The skin incision is curved, following the inner angle of the orbit and extending no farther than the supraorbital notch and downward on the nasal process of the maxillary bone. Usually, the incision is from 4 to 7 cm. in length. If, as in the case of extremely large sinuses, more room is needed, another incision can be made from the outer margin of the supraorbital notch to the outer canthus. I have never had to use the second incision.

The elevation of the periosteum exposes the anteroinferior wall of the frontal sinus, the frontal process of the maxillary bone, and a portion of the nasal bone. The nasal fissure that joins the glabella to the nasal bone and nasal process of the maxillary bone can be seen. The lacrimal sac is now raised from its bed, and the periosteum of the lamina papyracea is separated for a few millimeters. The anterior ethmoid artery should be identified, tied and cut. I have found that a large, round electrically driven burr is best for uncovering the mucoperiosteal membrane beneath the nasal process of the maxillary bone and the nasal bone. The membrane is then separated, a few millimeters at a time, with some type of nasal periosteal elevator. The bone is removed with rongeurs, and the flap is uncovered.

The membrane is separated from the overlying bone by passing variously curved elevators downward as well as laterally and upward to the nasal fissure in order to obtain as large a flap as possible and enough to cover the inside of the glabella. After enough of the bone has been removed to make a large flap, two vertical incisions are made downward from the nasal fissure and separated enough to make a flap of the proper width. The horizontal or lower end of the flap can now be cut, either by going through the opening or, if necessary, by going through the nostril. This completes the flap. This sturdy piece of mucoperiosteum, from 1 to 4 cm. in width and from 3 to 7 cm. in length, is to be turned upward to line the medial wall of the new nasofrontal opening and part of the frontal sinus.

From the University of Oregon Medical School, Portland.

Presented at the Sixty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 14-19, 1956, Chicago.

This flap will cover the inside of the glabella and prevent the formation of granulation and resultant adhesions which could cause closure of the new frontal nasal opening. At the same time, it will extend into the sinus, and epithelium will grow from it, covering the granulations that are formed within the sinus cavity.

The flap is now pushed to the front of the nose and protected by a gauze sponge while work on the frontal sinus, the ethmoid cells, and the sphenoid is completed.

Again using the burr, the innermost portion of the floor of the frontal sinus is exposed. The entire floor of the sinus is removed, leaving absolutely smooth edges. The entire membrane of the frontal sinus is carefully removed by separating it from the bone without curetting. I believe that the curet should never be used in removing sinus membrane. Following this, the glabella is prepared for the flap by carefully removing all the cancellous bone. This bone is an ideal place for the formation of exuberant granulations and would consequently close the newly made duct or nasal opening. The diseased ethmoid sinuses and the lamina papyracea, under complete visualization and accessible in any direction back to the sphenoid wall, are now carefully removed. If necessary, the sphenoid is exenterated, for all sinus infection must be reduced to a minimum.

The mucoperiosteal flap, which has been pushed aside, is now turned upward to line the medial wall of the new frontal opening and is kept in place either by a cigarette drain or by a soft rubber tube extending into the nose. The ethmoid and sphenoid regions are loosely packed with one-half inch gauze saturated with cod liver oil. The skin incision is closed by a few interrupted silk or linen sutures. Some surgeons prefer to bring the periosteum edges together with catgut sutures and then close the skin incision. That part of the gauze packing which comes away easily is removed about the third day. The tube holding the mucoperiosteal flap in place, and the skin sutures are removed the fourth or fifth day. The gauze packing should never touch the flap because it is apt to adhere and pull the flap out of place. If the flap is well made and sufficiently large, the aftercare is very simple and the new frontal opening will stay open permanently. I never irrigate; let the sinuses recover without irrigation. Crusts and discharge may be removed by careful spot suction.

The success of this operation depends on (1) the removal of the entire floor, leaving no projecting shoulders or spicules of bone; (2) the removal of every particle of the mucosal lining; (3) the removal of the diseased ethmoid cells and, if necessary, the sphenoid; (4) the removal of the cancellous bone of the glabella; and (5) the making of a mucoperiosteal flap of proper size.

RESULTS

In 1952, I reported fifty-seven chronic frontal sinus operations performed by this method and stated that I had had no failures, at least none that I had been able to locate.[1] Since then, the staff of the University of Oregon Medical School and I have operated upon forty more patients with chronic frontal sinusitis by this method and still there are no failures. I am sure that sooner or later there will be a failure, because no operative procedure is 100 per cent perfect. This procedure may be followed for patients who have been operated upon previously by the original Lynch or Sewall technique. Of the forty patients operated upon since 1952, three had been operated upon previously, but had had recurrences. Two years before coming to us, one patient had had an intranasal frontal sinus operation, or an

attempted enlargement of the duct by rasping, which in my opinion should never be done, and three external operations for a fistula which drained periodically. Up to the present time he has had no more trouble. Another one of the three, a boy 11 years old, had one external operation but still had a discharging frontal sinus. In none of these three patients had the membrane lining the sinus been properly removed, nor had the entire frontal sinus floor been smoothly removed. Another patient, a woman, had had several nasal operations, including an external one in which the superior and middle turbinates were removed. We operated upon her three years ago. When I made the flap, it was the thickest, toughest membrane that I had ever encountered. It measured 1 cm. in thickness. It had to be thinned before I was able to put or keep it in place. To thin it, I removed the periosteum and part of the overlying structures, leaving practically only the mucous membrane. I was sure that this operation would fail, but the patient has been symptom free up to this time, even though the frontal opening is very small.

McNally,[2] in 1954, reviewed three hundred cases operated upon at the Montreal General Hospital by the various staff members. One case, which was particularly interesting to me, was that of a patient who had had a right and left frontal operaton. A mucoperiosteal flap was made on one side but not on the other. In two years a recurrence necessitated a second operation on the side on which no flap was made; the side with the flap needed no further operation.

OTHER OPINIONS

Before starting this paper I wrote to six prominent otolaryngologists in different parts of the country whom, I knew, had been doing this operation the past few years. Their comments are interesting. One answered:

> I have done only two cases using the mucoperiosteal flap, but the results were good in both cases.

Another wrote:

> I can recall using the frontal mucoperiosteal flap in three patients since becoming acquainted with it. None of these have had a subsequent recurrence of their difficulty, as far as I know. In my opinion the use of the mucoperiosteal flap is the operation of choice.

Another wrote:

> We have had seven occasions to use the mucoperiosteal flap. In each instance the results have been good and there has been no necessity for further surgery in any case. We are exceedingly happy with the procedure. Today we approach a patient with fair confidence that we are in the process of solving his problem. This is in contradistinction to my feeling of "When next" when I did the procedures of "bore open and try to keep open."

Another wrote:

> As best we can figure, we have done ten or twelve of these cases, and have had no bad results whatsoever. As a matter of fact, I am highly enthusiastic about this technique, and highly recommend it to everyone.

Another wrote:

> Since 1952 we have done thirty external frontal sinus operations, and in sixteen of them we have used your flap. In most of the other cases the operation was for some condition such as osteoma, in which we were able to preserve a normal mucosal-lined nasofrontal duct. In one or two an ablating operation was done. We have not had a recurrence as yet in any case in which we used the Boyden flap. I feel this is the most satisfactory type of operation on the frontal sinus. . . .

The last one has stopped doing nose and throat work, so had not used the procedure.

All of these men stated that there were few frontal operations to do in the past few years. However, sixty-eight patients have been operated upon since 1952 in six localities of this country. Realizing the number of otolaryngologists in this country and in Canada, we can readily

see that there are still many patients with chronic frontal sinusitis, and all otolaryngologists should be able to help them.

CONCLUSIONS

After twenty years' experience in using the mucoperiosteal flap, I see no reason for doing a collapse of the frontal sinus unless there is osteomyelitis present; nor do I advise the use of tantalum, acrylic tubes or any other foreign body to keep the duct open. Of course, a tube is worn for four to six days to give the mucoperiosteal flap time to adhere.

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ESTIMATION OF LOSS OF VISUAL EFFICIENCY

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THREE years ago the Council on Industrial Health of the American Medical Association, through the Joint Committee on Industrial Ophthalmology of the American Medical Association and the American Academy of Ophthalmology and Otolaryngology, expressed its dissatisfaction with the regulations and recommendations then in force, which were set forth in the American Medical Association's publication "Appraisal of Loss of Visual Efficiency," revised and adopted in 1940. The Council on Industrial Health was aware of some of the obsolete aspects of this earlier appraisal and of the possibility, if not certainty, of different opinions and different conclusions which might now be acceptable to industrial medicine, to industrial health commissions, to compensation boards, and to insurance companies.

The essayist, as a member of the Joint Committee on Industrial Ophthalmology, was requested to present this subject to the International Association of Industrial Accident Boards and Commissions for preliminary discussion; this was done on January 6, 1953. The Council on Industrial Health was aware of the widespread authoritative use of this appraisal by the various state commissions connected with the ophthalmological aspect of industry. At the same time, this Council was properly concerned over the fact that Superior Court decisions of dubious medical propriety had been rendered in various states. They were also aware that these decisions were not consistent and that, in some instances, they were at variance with the recommendations in force within the 1940 edition. Various unwarranted awards made were, in fact, due to the obsolete factors within the 1940 edition.

Following the meeting of the International Association of Industrial Accident Boards and Commissions, an independent committee was appointed by the Council on Industrial Health to revise this 1940 "Appraisal of Loss of Visual Efficiency" and to present the revision, when completed, to the House of Delegates of the American Medical Association, through the Executive Committee of the Section on Ophthalmology and through the regular meeting of the Section of Ophthalmology of the American Medical Association. The committee which was appointed consisted of Dr. F. Bruce Fralick, Dr. William F. Hughes, Jr., and myself. Dr. Hughes served as Secretary of this subcommittee. At this time, it is quite proper for Dr. Fralick and myself to express to Dr. Hughes our sincerest thanks and appreciation for assuming, with the mildest of protests, a secretarial task which turned out to be tremendous and which was carried out and completed with consummate skill.

This report was presented in its first tentative draft to the Executive Committee of the Section on Ophthalmology of the American Medical Association in 1954 for their discussion, criticisms, and recommendations. It was returned to the committee with recommended changes. In 1955, at the Atlantic City meeting, the committee's report was presented in its final draft, as a codex juris on the subject, under a new title, "Estimation

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of Loss of Visual Efficiency." This was approved and accepted by the American Medical Association at the 1955 general meeting. The enthusiasm from interested and involved organizations and commissions since its publication seems to confirm wholly the earlier opinions of the Council on Industrial Health as to the necessity for a revision.

It is manifestly impossible to present this report today in any satisfactory condensation or in abstract. The presentation to you today will call your attention to the major recommendations of the committee and will, at the same time, permit the discusser, Mr. Percival E. Thompson, a Chicago lawyer with wide experience in personal injury court procedures, to agree, to disagree, and to present the legal aspects, as these are seen from his standpoint. It is hardly necessary to say that the committee, in working over these revisions, has repeatedly sought legal advice as to phraseology; even as to the limits for recommendations upon a medical aspect, as these might be complicated by the legal aspects. One such instance is that portion of the revision which has to do with the estimation of visual acuity following operative aphakia. Regardless of how perfect the visual efficiency might be in such a situation, the fact still stands—the patient has lost a portion of an organ, i.e., the lens of an eyeball; hence it appears that he is entitled to some compensation for this.

The report is presented in eight subsections and in two major subdivisions.

The first four subsections, constituting the first major subdivision, are (a) essential factors to be considered in determining visual efficiency; (b) the determination of central visual acuity for both distance and near; (c) the estimation of the efficiency of an impaired field of vision; and (d) the determination of a percentage loss of efficiency as the result of disturbed ocular motility in one or both eyes. The second subdivision of the report is concerned with the calculations for the estimation of the visual efficiency of one eye, and similarly of both eyes, as this efficiency is concerned with corrected visual acuity for distance and near, with the extent of the fields of vision, and with the state of the ocular motility as related to the degree of binocular single vision present. The last two brief sections of the revision present types of ocular abnormalities not to be included in visual efficiency factors and certain rather important recommendations relative to the period when a case shall be considered at maximum visual efficiency, then evaluated as to the estimation of loss of visual efficiency.

Some of the major changes are as follows:

When determining central visual acuity, the best vision obtainable with ophthalmic lenses should be used to determine the degree of central visual acuity. This is quite at variance with the rulings of some state commissions, wherein visual acuity is determined without the use of correcting lenses. In some instances, the use of contact lenses might further improve vision reduced by corneal disease or injury; however, the practical difficulties of fitting, the expense, and the tolerance to contact lenses are sufficiently important, at the present time, to favor the recommendation that only regular ophthalmic lenses be used to determine the best corrected vision. The visual handicap resulting from aphakia corrected by regular ophthalmic lenses can only be estimated. However, even uncorrected aphakia should not be considered equivalent to a blind eye, because of the usefulness of side vision and the possibility of correction if the opposite phakic eye should be injured subsequently. The practice of consider-

ing monocular aphakia as the full loss of visual efficiency in one eye does not recognize the value of an eye with no abnormality except the loss of the lens. With these considerations in mind, it is recommended that monocular aphakia be considered as a partial visual disability, viz., 50 per cent of the central visual efficiency of that eye when corrected by lenses. For example, if corrected vision in the aphakic eye is 20/40, the allowable central visual efficiency of that eye would be 0.50×0.85 (43 per cent), indicating a loss of central vision of 57 per cent. In cases in which the patient's industrial activities require use of the corrected aphakic vision, whether this be corrected with ophthalmic lenses or with a contact lens, it is recommended that this state of aphakia be considered an additional partial visual handicap of 25 per cent of the best corrected binocular visual efficiency, as this is calculated in Section 6 of the report. For example, if the binocular visual efficiency of the patient with corrected aphakic vision in one eye is 75 per cent, the final estimation of the visual efficiency of that patient would be 0.75×0.75, or 56 per cent.

In general, the committee felt that the table set up by the 1925 and the 1940 American Medical Association committees overestimated the loss of visual efficiency for distance acuities poorer than 20/200, and underestimated the loss of near acuities poorer than 14/28, which is equivalent to Jaeger 3, or six-point type.

A visual acuity of 20/20 or better (6/6 or better, or visual angle of 0.1 minute or less) is considered perfect acuity for distance vision; and Snellen 14/14, Jaeger 1, or three-point type (with presbyopic correction if necessary) is considered perfect acuity for near vision (14 in., or 36 cm.). A revision of the estimations of percentage loss of visual acuity corresponding to the Snellen and visual-angle notations for distance and near is given in tables I and II. This has been done in the light of statistical reports of average "normal" vision, and of experience in the disabilities caused by reduced visual acuity. No scientific studies are available relating central visual acuity, visual field, and motility deficiencies to the actual performance of various duties.

Also, the visual disability in reading with acuity for near of 14/35 (Jaeger six-point or eight-point type) is far greater than the visual disability for distance with 20/50 vision, which has the same visual angle. The revised tables I and II represent an effort to relate visual acuity and visual disability more closely, in the light of experience.

Because of the importance of good near vision in the performance of work, the revised limits of acuity weight this disability more heavily than the 1940 American Medical Association table did. Calculation of the percentage of central visual efficiency for both distance and near vision would, therefore, be the average of the two percentages.

In estimating the state of fields of vision, the isopters recommended are about 10 to 15 degrees less than the average normal, thus allowing for poor or delayed subjective responses and for undue prominence of the brow and the nose. Figure 1 illustrates the committee's recommendation as to a normal field of vision.

The estimation of total visual efficiency is related to the function of the extraocular muscles in two major ways: the absence of diplopia, and the presence or absence of binocular single vision. It is doubtful, except on looking downward, whether diplopia causes significant visual difficulty unless it is present within 30 degrees of the center of fixation. This basic premise is one of the biggest differences of opinion between

TABLE I

PERCENTAGE OF CENTRAL VISUAL EFFICIENCY CORRESPONDING TO CENTRAL VISUAL ACUITY NOTATIONS FOR DISTANCE

SNELLEN		VISUAL ANGLE, IN MINUTES*	PER CENT CENTRAL VISUAL EFFICIENCY	PER CENT LOSS OF CENTRAL VISION
ENGLISH	METRIC			
20/16	6/ 5	0.80	100	0
20/20	6/ 6	1.00	100†	0
20/25	6/ 7.5	1.25	95	5
20/32	6/10	1.6	90	10
20/40	6/12	2.0	85	15
20/50	6/15	2.5	75	25
20/64	6/20	3.2	65	35
20/80	6/24	4.0	60	40
20/100	6/30	5.0	50	50
20/125	6/38	6.3	40	60
20/160	6/48	8.0	30	70
20/200	6/60	10.0	20	80
20/300	6/90	15.0	15	85
20/400	6/120	20.0	10	90
20/800	6/240	40.0	5	95

*Denoting each constituent part of test character. When the visual angle of each constituent part is multiplied by 5, the product is the visual angle of entire test character.

†For purposes of calculation of the per cent of central visual efficiency, 100% is used. This does not consider the fact that the average normal person usually has a visual acuity of 20/16 corrected by ophthalmic lenses.

TABLE II

PERCENTAGE OF CENTRAL VISUAL EFFICIENCY CORRESPONDING TO CENTRAL VISUAL ACUITY NOTATIONS FOR NEAR

SNELLEN	JAEGER	POINT	VISUAL ANGLE IN MINUTES*	VISUAL EFFICIENCY	PER CENT LOSS OF CENTRAL VISION
14/14	1—	3	1.0	100	0
14/18	2—	4	1.25	100	0
14/22	. . .	5	1.6	95	5
14/28	3	6	2.0	90	10
14/35	6	8	2.5	50	50
14/45	7—	9+	3.2	40	60
14/56	8	12	4.0	20	80
14/70	11	14	5.0	15	85
14/87	. . .	. .	6.3	10	90
14/112	14—	22	8.0	5	95
14/140	. . .	. .	10.0	2	98

*Denoting each constituent part of test character. When the visual angle of each constituent part is multiplied by 5, the product is the visual angle of the entire test character.

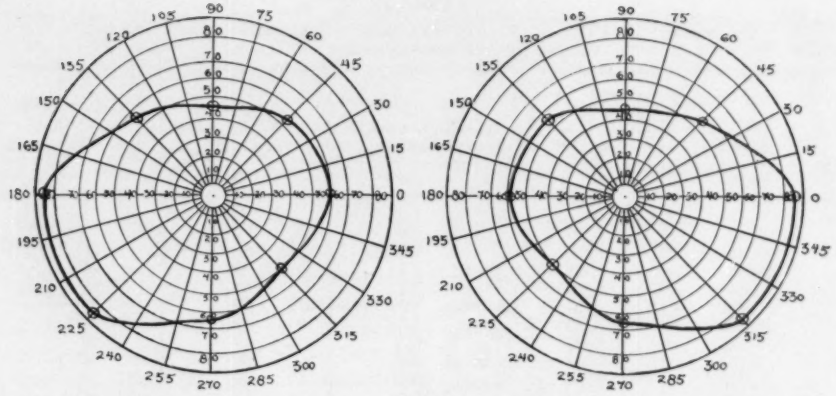

FIG. 1—Recommended normal field of vision. White field; 3/330 stimulus. (From A.M.A. Arch. Indust. Health, 12:439-449 [Oct.] 1955.)

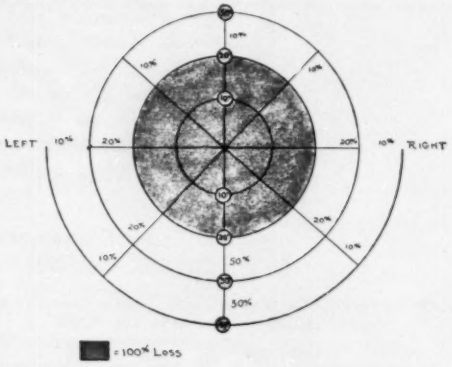

FIG. 2—Percentage loss of ocular motility of one eye in diplopia fields. (From A.M.A. Arch Indust. Health, 12:439-449 [Oct.] 1955.)

the present committee's recommendations and those earlier recommendations in force prior to this revision. Figure 2 illustrates the percentage loss of ocular motility in one eye because of diplopia. The shaded area constitutes complete loss of motility for one eye. The other areas are to be considered separately. The percentage of disability from lost ocular motility is based upon the diplopia in three meridians: above—10, 20, and 30 degrees from the straight ahead position; horizontally; and below—10, 20, 30, and 40 degrees away from the straight ahead position.

If a patient has lost binocular single

vision as a result of disease or injury and because of suppression and is free of diplopia, the degree of visual disability will depend upon the importance of good depth perception in the performance of the patient's work. The demands for depth perception vary greatly. However, loss of binocular single vision without other ocular abnormalities probably does not represent more than a 50 per cent loss of efficiency in ocular motility of one eye in the average case. In this instance, in agreement with the previous committee's recommendations, a more nearly correct estimation of visual efficiency is obtained by the following computations: multiply the percentage of efficiency of the better eye by three; add this result to the percentage of efficiency of the poorer eye; divide this sum by four. The result represents the degree of binocular visual efficiency for the two eyes. In this estimation, also, all cases with binocular visual efficiency of less than 10 per cent are to be considered as blind for all practical, industrial, and legal purposes.

CONCLUSIONS

Although such a recommendation was not incorporated in the committee's report, it is the hope of the committee and of the parent organization responsible for the committee's investigation, that is, the Council on Industrial Health of the American Medical Association, that this report will again become the basis for uniformity in the standards set up by various state commissions. There is no reason why the practices and statutes of one state regarding industrial injuries and their effects on visual efficiency should be at great variance with those in force in another state for the same lesion, the same loss of efficiency, or the same degree of disability.

There is much chaos present today in the coordination of medicine, law, and social economy. Calculated sentiment, political expediency, inept medical testimony, and conflicting legal attitudes are responsible for part of this. Organized medicine can help to correct abuses and assist in just awards. This recent revision has been made with that aim.

LEGAL ASPECTS OF THE AMERICAN MEDICAL ASSOCIATION'S REPORT ON ESTIMATION OF LOSS OF VISUAL EFFICIENCY

PERCIVAL E. THOMPSON
CHICAGO, ILLINOIS

BY INVITATION

THE work product of your Committee, of which Dr. Spaeth has given you a brief summary, is an attempt on the part of ophthalmologists, to arrive at a formula, or rather a series of formulas, by which they may determine, if not with greater accuracy, certainly with greater uniformity of opinion than previously, the loss of visual efficiency which a given individual may have sustained through accident or disease. Uniformity of medical opinion as to the percentage or proportion of loss of visual efficiency is a highly desirable objective in the field of industrial medicine insofar as that field overlaps the legal field of compensation for industrial disease or for industrial injury to the eyes or to their accessory organs.

At the outset, let me make myself clear. What I am about to say applies only to industrial diseases and accidents —to the so-called Workmen's Compensation cases. If I may, in order to show why this uniformity of medical opinion is desirable, I would like to discuss very briefly with you the philosophy behind the various workmen's compensation statutes of the different States of the Union. At the present time forty-seven of the forty-eight states have workmen's compensation acts.[1] Perhaps about one half of the normal working population of some 60 million persons in this country are covered by these acts. Workmen's compensation insurance is by far the largest form of casualty insurance in the United States, and the total amount expended by American employers for premiums to private insurance companies, state funds, and self-insurance plans probably exceeds 1500 million dollars annually.[2] A substantial portion of this amount of money is returned to the dependents of approximately 20,000 workers who are killed annually in industry, to the 100,000 workers who are seriously or permanently injured, and to many others who are injured but fortunately recover after varying periods of time.

Workmen's compensation made its appearance in this country shortly after the turn of the century. It came about as a sort of social revolution or social reform against the legal system of compensation for employees injured in industry which had theretofore been in vogue in the courts of this country. Under that prior system, an employee who lost an arm or a leg or his eyesight could recover compensation for his injury from his employer only in the event that he could prove that that injury was caused by his employer's carelessness or what we lawyers call "negligence." Even then, if the injury was partially the employee's fault, or if it was caused by a fellow employee, or if the injured employee had known of the danger inherent in his job and had "assumed the risk" incident to his employment, he could not recover. It has been estimated that under that system about 80 per cent

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1. Mississippi alone has no Workmen's Compensation Act.

2. 17 NACCA Law Journal, p. 34.

of the cases which were filed were lost,[3] and the seriously injured or maimed workman was relegated to the charity of relatives or friends, or became a public charge in the county poor house. If he was killed in the course of his employment, because of the difficulties of proof his widow and children had even less chance of receiving compensation than would the employee himself, had he lived.

When it was first proposed in this country that industry should add to the price of its products the cost of furnishing medical attention and weekly awards of sufficient amounts to allow its injured employees to exist until such time as they were rehabilitated, and to pay specific sums for certain specific injuries, irrespective of the questions of negligence, contributory negligence, assumed risk, and all of the other old common-law folderol, the proposal met with violent opposition on all sides. Industry feared the cost; insurance companies who had been receiving large premiums and paying small losses under existing laws feared the loss of profits; and peculiarly enough, the workers themselves, through their unions, in many instances opposed the passage of the original Workmen's Compensation Acts. Nevertheless, over a period of several decades the basic soundness of some plan requiring industry to make some provision for the human carnage it leaves in its wake, rather than foisting the consequent economic burden and loss of that carnage upon the public at large, became so apparent that today workmen's compensation, as we now know it, is almost universally accepted.

While the acts of the different states vary in detail, most of them provide for medical service to be furnished to an injured employee; in addition, a weekly amount is paid to the workman for temporary disability during the time he is unable to work. This weekly payment is generally not large, but theoretically, at least, this compensation is an amount upon which he and his dependents can live during his period of convalescence. In cases of permanently disabling injuries, which will or may affect either the workman's ability to carry on his work or to obtain employment, specific awards are frequently provided. Thus, when a workman has lost an arm or a leg, or even his eyesight, there is generally no problem in the administration of the Act, as the Act itself generally provides a specific sum for these specific losses. Much more common, however, than the complete loss of a member is the permanent partial loss of the use of that member. Here again there should be no problem, as it is usually provided that in this case the employee is entitled to the same proportion of the total award for a specific loss as the percentage of loss of use of the injured member bears to the total loss of that member; in other words, if an employee has permanently lost 50 per cent of the use of a leg, he would be entitled to 50 per cent of the amount that would have been awarded had his leg been amputated.

Now, in theory, this is all very well—but just what is the loss of 50 per cent of the use of a leg? What functions must be missing and what functions must remain so that a doctor may conscientiously testify that his patient has lost no more and no less than 50 per cent, or 25 per cent, or 75 per cent of the use of a leg? Even more perplexing is the problem facing the practitioners in the field of ophthalmology. Ophthalmologists rarely differ radically, at least upon the end result, as to the physical injury to an eye or even as to the effects of that injury upon the patient's ability to see. However, ophthalmologists have, in the past, differed widely in their opin-

3. Horovitz: Injury and Death under Workmen's Compensation Laws, p. 3.

ion as to the percentage of loss of visual efficiency due to a given condition. This is understandable. Individual practitioners might be, and have been, at wide variance in their opinion as to the percentage of loss of vision a given individual has sustained by reason of injuries which affect, not only the ability of the patient to see the characters upon a chart, but to utilize the entire normal visual field. In a large part, these differences of opinion reflect only the individual practitioner's personal opinion as to the importance, or lack of importance, of the loss of a given function of the eyes. The report of your Committee has the virtue of laying down a standard by which all capable practitioners, after examining a patient who has been injured at his place of employment, will arrive at substantially the same result, within reasonable limits, for the same injury by the application of the formulae suggested.

I have been asked by Dr. Spaeth to criticize the Committee's report and I am going to be foolhardy enough to do just that. First, let me say this: I shall not criticize the methods of arriving at the estimation of loss of visual efficiency, either insofar as those methods involve medical techniques or involve the formula by which you arrive at the end result. Those are problems which must be resolved by ophthalmologists, rather than by lawyers. My first criticism is not a criticism at all but rather a plea, addressed not only to ophthalmologists but to the entire medical profession, and it is this: Is there not some method by which doctors can adopt a vocabulary of descriptive words and terms which can be more readily understood by lawyers, judges, and other laymen? We in the legal profession have in the last twenty years endeavored to abolish the legal jargon consisting of Latin and Greek phrases, although I frankly admit that the movement has met with great opposition. If a doctor is to be understood in court, or before an industrial commission, he must use language which can be understood if he is to do justice to his patient's claim. Recently the Supreme Court of Minnesota had this to say in one of its opinions:

> The present issue, whether Susnik's accident contributed to his death nearly four years later, has required our consideration of the large volume of testimony, which conflicting notions of the doctors (there were only 18 of them) leave in a mass of irreconcilable contradiction. With the aid of a medical dictionary we have striven to understand, not only the views of each of them, but the factual basis assigned by each for his conclusion.[4]

Recently a doctor was good enough to furnish me with a medical report describing the injuries sustained by a client of mine. Part of that report read as follows:

> On the inferior aspect of his right gluteus maximus immediately superior to the infragluteal sulcus extending laterally to the ischial tuberosity there was an area of ecchymosis and tumefaction in the form of hematoma measuring 7.5 cm. by 8 cm. with a total estimated volume of 75 cc.

How much simpler it would be to say that this patient had a black and blue mark on his hip. I will spare you the rest of the good doctor's report.

My second criticism of your Committee's report is that it contemplates determining loss of visual efficiency, in part at least, after the claimant has been fitted with corrective lenses. I doubt the propriety and the legality of this method of determination. After all, most of these acts provide very modest awards for specific losses.[5] No court, to my knowl-

4. Susnik vs. Oliver Iron Mining Co., 205 Minn. 325 @328.

5. Compare the benefits under the Illinois act for the loss of an eye (Ch. 48, Sec. 138.8-13 Ill. Rev. Stat. 1955) amounting to less than $6,000 to the award of a jury in the Cook County Circuit Court of $50,000 for the same injury in a non-compensation case (Petrakis vs. Pepsi Cola Bottling Co., case 47C1758).

edge, has held that fitting a patient who has lost a hand with a prosthesis should diminish the specific award provided for by statute for the amputated hand. Neither am I aware of any court decision which holds that providing a workman who has lost the major portion of his hearing with a hearing aid, equipped with fully charged batteries, would be a discharge in whole, or in part, of the employer's obligation. With regard to an employer seeking to discharge his obligation, under the Missouri Compensation Act, by furnishing an employee with false teeth, a Missouri court had this to say:

> The loss of 31 teeth is a serious handicap to any one. It is a severe mutilation and permanent disfigurement. . . . No one could be so devoted to the practical and artificial as to claim for 'store teeth' equal advantage with the sound and natural incisors and molars. If there be such a one, we apprehend that time holds for him certain and complete disillusionment.[6]

While there is some conflict of judicial opinion, the greater weight of legal thought upon the matter has been to the effect that the loss of visual efficiency, sustained by reason of an industrial accident, should be determined on the basis of the eye as it is, and without the use of corrective lenses.[7]

6. Betz vs. Columbia Telephone Co., 224 Mo. App. 1004.
7. See collection of cases in 99 A.L.R. 1507.

EARLY TREATMENT OF ORBITAL FLOOR FRACTURES

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TRAUMATIC comminution of the facial bones has increased because of the acceleration in the traveling speed of motor vehicles. Complications such as enophthalmos and diplopia are encountered more frequently in fractures involving the zygoma and maxilla and the floor of the orbit. These complications have led us to reconsider some of the surgical aspects of these comminuted fractures and, in particular, their early treatment.

The orbital contents are protected by a ring of strong bony abutments, the supraorbital arch of the frontal bone, above; the zygoma, laterally; the frontal process of the maxilla, medially; and inferiorly, the relatively thick rim of the orbital floor formed by the zygoma and the maxilla. Situated between two strong abutments, the zygoma and the frontal process of the maxilla, the orbital rim is supported below by relatively thin bone which constitutes the anterior wall of the maxillary sinus. Posterior to the rim, the orbital floor consists of thin bone which separates the orbital cavity from the maxillary sinus; this thin portion of the orbital floor is weakened further by the infraorbital groove or canal. The orbital walls are thin in two additional areas, over the temporal fossa laterally and the lamina papyracea of the ethmoid medially. These thin areas are readily demonstrated by transillumination of the dried skull.

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In fractures of the zygoma and maxilla with marked displacement of the orbital rim, one may anticipate concomitant fracture of the thin orbital floor. In fractures with slight displacement, however, the involvement of the weak areas of thin bone in the orbital floor is not obvious. This has been demonstrated to us in observations made during operative procedures to restore the orbital floor by bone grafting following malunited fractures.[1,2,3,4] Depression of the thin portion of the orbital floor was noted and in some cases a direct opening between the orbit and the maxillary sinus was seen.

These observations lead us to believe that comminuted fractures of the thin portion of the orbital floor can occur by two different mechanisms: (1) backward displacement of the strong bone forming the orbital rim, which results in comminution of the thin bone; (2) increased internal orbital pressure caused by a blow on the soft tissues of the orbit, producing fractures of the weak area without affecting the more resistant orbital rim, a type of fracture which we refer to as the "blow-out" fracture. This latter type of fracture, less frequent than the former, may be caused by large, nonpenetrating objects such as the human fist, a baseball, a cricket ball; in one of our case histories the striking object was a ball used in the Irish game of hurling.[4] Smith and Regan[7] have reproduced this type of "blow-out" fracture experimentally on the cadaver.

Gravity causes a downward displacement of the orbital contents into the maxillary sinus when the floor of the

orbit is comminuted. Additional displacement is caused by accumulation of blood and edematous fluid in the injured periorbital tissues, and later during the healing period by the pull of maturing scar tissue. The numerous small pieces of bone remain connected through the intermediary soft tissues, and the floor sags hammock-like into the maxillary sinus; if damage is severe enough, the continuity of the floor is interrupted and orbital fat escapes into the maxillary sinus.

The result of this anatomical disturbance is a downward displacement of the orbital contents and an enlargement of the orbital cavity. Enophthalmos and diplopia are frequent complications of such fractures.

TRAUMATIC ENOPHTHALMOS

Enophthalmos appears insidiously, usually after the subsidence of the edema, hematoma and inflammatory reaction accompanying the fracture. The eyeball sinks inward progressively, and once established, enophthalmos is difficult to correct because of atrophy and shortening of the intraorbital structures, thus constituting a serious deformity in an otherwise satisfactorily rehabilitated patient. Because of this fact, an understanding of the mechanism of production is important.

Lower animal forms possess a muscle, the orbitalis muscle, which spans the floor of the orbit, covering the inferior orbital fissure; this muscle protrudes the eyeball for purposes of focusing vision. In man a vestige of this muscle is also designated as the orbitalis muscle. Some anatomists discount its importance; others claim that the paralysis of this muscle, innervated by the sympathetic nervous system, is a factor in the production of enophthalmos in conditions such as Horner's syndrome. Others feel that atrophic changes in the orbital fat due to injury of the sympathetic innervation may be responsible for the production of enophthalmos. Still other factors have been held responsible for traumatic enophthalmos: dislocation of the trochlea of the superior oblique muscle, cicatricial contraction of retrobulbar tissue, and rupture of the orbital ligaments or of fascial bands.

Mechanical factors appear to play an important role in orbital fractures; among these are the escape of fat into the maxillary sinus, frequently seen in comminuted fractures involving the floor of the orbit, and the enlargement of the orbital cavity due to the displacement by fracture of the orbital walls.[5] Lang contended that a fracture occurred in every case of enophthalmos and stated:

> I suggest that the injury may have produced a fracture and a depression of a portion of the orbital wall; the orbital fat would then be no longer sufficient in quantity to fill this enlarged postocular area without a sinking-in of the globe from atmospheric pressure and a resulting limitation in ocular movements.

Pfeiffer[6] reviewed 120 cases of fracture of the bones of the face with orbital involvement and found that enophthalmos was present or had developed subsequent to injury in fifty-three of these cases. Fracture of the orbit was noted in every case of traumatic enophthalmos. In many of the cases fracture of the orbit was obvious, but in a number of cases the fracture was internal or deep in the orbit, unassociated with solution of continuity or deformity of the margin of the orbit, and was detected only through roentgenograms. In a number of cases the backward displacement of the eye was as much as 9 mm.; in most cases, however, the posterior displacement was 3 or 4 mm. The displacement of the eye would have been overlooked clinically in a number of Pfeiffer's cases

if exophthalmometry had not been employed following radiologic findings of fracture.

The radiographic findings were characteristic. In every case the floor of the orbit was broken through into the maxillary sinus below. Not only were fragments seen frequently in the maxillary sinus, but increased density was demonstrable in the maxillary sinus, due to the presence of orbital tissue. In severe enophthalmos the maxillary sinus was filled with orbital tissue.

It has been suggested that posterior displacement of the orbital contents caused by trauma is responsible for an internal orbital fracture. The force of the blow received by the eyeball is transmitted to the walls, producing fracture of the more delicate portions. The floor is usually involved because the thin posterior convex portion of the floor bulges upward posterior to the eyeball, thus being in a position to receive most of the force transmitted by the eye. Fracture of the lamina papyracea is sometimes observed. These fractures tend to increase the size of the orbit.

We believe that traumatic enophthalmos is due to an enlargement of the orbital cavity; the orbital fat distributed in a larger cavity no longer suffices to maintain the normal protrusion of the eyeball (fig. 1). In some cases the orbital contents may be held backward by scar tissue or by herniation and incarceration of orbital tissues between depressed bony fragments. The reverse mechanism, diminution of the size of the orbital cavity by backwardly displaced overlapping fragments, accounts for traumatic exophthalmos, an apparently anomalous condition occasionally seen following fractures. Orbital hematoma is another cause of traumatic exophthalmos. True exophthalmos must be differentiated from pseudoexophthalmos which is due to a backward displacement of the orbital rim. Increase in the vertical width of the affected interpalpebral fissure may give a false impression of exophthalmos.

Determination of the degree of enophthalmos is based upon the relative position of a line projected from the lateral orbital margin to the profile of the zenith of the cornea. In the case of loss of bone and osseous malposition, fixed points on the affected and unaffected sides cannot be used in diagnosis. In such cases, the degree of ocular protrusion or recession is measured by the difference in the number of millimeters required to focus an instrument on each cornea, with the patient's head and the focusing instrument placed in a central position. This measurement may be calculated with a corneal microscope and head rest or by means of an adapted keratometer. The calibration of either instrument for use as an exophthalmometer is not a difficult procedure.

TRAUMATIC DIPLOPIA

Diplopia occurs in comminuted fractures of the orbital floor with downward displacement because the visual axis of the affected eyeball is not in alignment with that of the opposite eye. The treatment of diplopia resulting from malunited fractures of the orbital floor by restoration of the floor by bone grafts, thus replacing the orbital contents to a suitable level, has been described in previous publications.[1,3,4]

It is necessary to replace the bone and wire the frontozygomatic suture line when a separation between zygoma and frontal bone results in downward displacement of the zygomatic portion of the floor of the orbit.

The orbital floor may be so markedly comminuted that the orbital contents are depressed into the maxillary sinus. In some cases, fragmented fracture of the

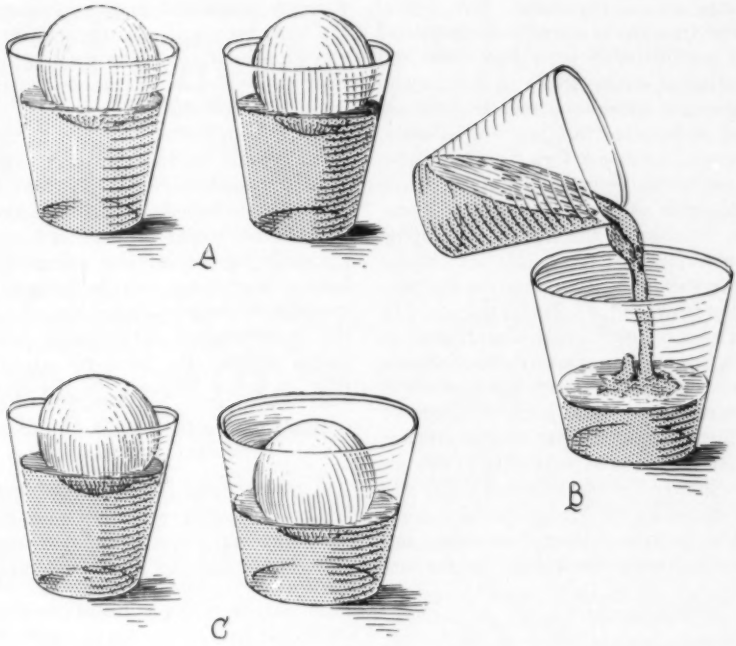

FIG. 1—Diagrammatic representation of one of the mechanisms of production of enophthalmos: enlargement of the orbital cavity. (A) The glass represents the orbital cavity, the water, the orbital fat and the ping-pong ball, the eyeball. An equal amount of water in each glass maintains the balls at the same level. (B) Water is poured from one glass into a glass with larger diameter. (C) The ball in the larger glass is at a lower level although the amount of water is equal to that in the smaller glass. Thus, the eyeball becomes enophthalmic not only when fat escapes from the orbit but also when the orbital fat is in an orbit enlarged by fracture. (From Converse, John Marquis & Smith, Byron.[4])

thin portion of the floor causes a slight depression of the orbital contents; the formation of scar tissue causes a further downward displacement of the eyeball. The periorbita may be pinched between sharp comminuted bone fragments; injury to the inferior rectus muscle is thus an additional cause of diplopia because of imbalanced function of the extraocular musculature.

Diplopia is obvious immediately after injury by the gross downward displacement of the orbital contents. In many cases its appearance is as insidious as that of enophthalmos, becoming manifest only after subsidence of edema and resorption of hematomas. The eyeball is downwardly displaced, the superior palpebral groove is accentuated, and upward rotation of the eyeball is difficult or impossible.

The pattern of diplopia in orbital floor fracture is variable since it is proportional to the degree of intraorbital tissue

damage and displacement. The typical severe fracture is usually accompanied by a restriction of both depression and elevation of the injured eye. Edema and ecchymosis within the muscle cone account in part for limitation of motility. Observations in our series of cases have led us to the conclusion that limitation of elevation and depression is due mainly to fixation of the tissues enveloping the inferior rectus and inferior oblique muscle sheaths by incarceration and herniation between the bone fragments. The inferior oblique muscle disturbance is less evident than that of the inferior rectus because the sheath involvement is so much closer to the point of origin of the muscle. The origin of the inferior oblique muscle is in proximity to the upper orifice of the nasolacrimal canal and is protected by the strong frontal process of the maxilla. Cover, uncover, and comitance tests could lead to the erroneous conclusion that involvement of the superior rectus muscle accounts for elevation limitation. The fixation of the inferior rectus muscle is demonstrated by the forced duction test, that is, by traction with forceps applied to the inferior rectus tendon. Surgical exploration of comminuted orbital floor fracture has convinced us that limitation of both elevation and depression is caused primarily by the inferior rectus disturbance. Rectifying this pathologic dysfunction by surgical means has alleviated the oculorotatory disturbance and diplopia without the need for an operation upon the superior rectus muscle.

SURGICAL MANAGEMENT

A comminuted fracture involving the floor of the orbit, whether or not diplopia is present, requires positive surgical measures if enophthalmos and diplopia

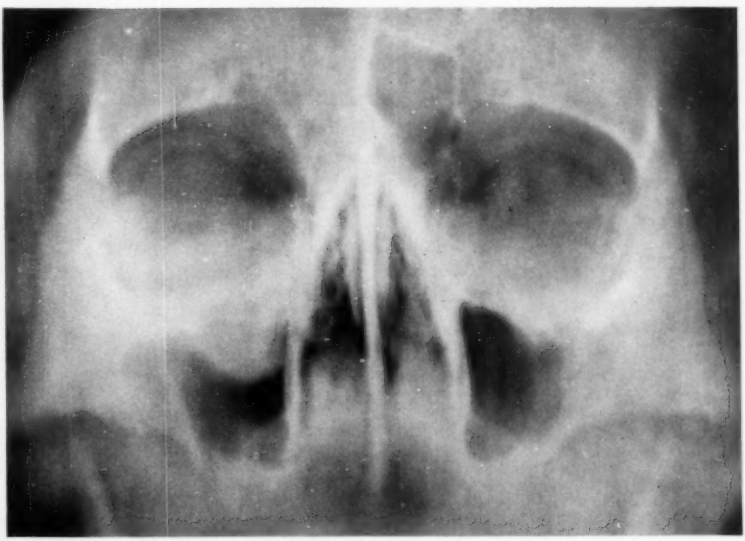

FIG. 2—Laminograph demonstrating the extrusion of the orbital contents into the maxillary sinus. Radiological examination had failed to reveal any abnormality of the orbital floor.

are to be relieved or prevented. Diagnosis of fracture and evaluation of displacement of fragments is relatively easy in fractures involving the zygoma and the rim of the orbital floor. Fractures of the weak portion of the orbital floor and the "blow-out" fracture without orbital rim involvement are more difficult to demonstrate by radiologic examination. Two positive means of diagnosis are available in these cases: (1) laminographic examination (fig. 2) which, in our opinion, should be routinely done in all orbital floor fractures and (2) direct examination of the orbital floor through the Caldwell-Luc approach to the maxillary sinus.

When there is evidence of extrusion of the orbital contents into the maxillary sinus, either through a rent in the weak portion of the orbital floor or as a result of a generalized sagging of the floor, continuity and correct level of the orbital floor are best restored by an iliac bone graft performed as soon after injury as possible.[4] This bone graft, suitably shaped, is introduced through an incision in the lower eyelid.

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DISCUSSION

D. F. FISHER, M.D., Memphis, Tenn.: I have enjoyed this fine paper by Dr. Smith and Dr. Converse. I am going to disagree with the method they have used, not in a spirit of criticism, but because I know a better means of handling these cases.

For a number of years my partner, Dr. D. H. Anthony, and I have been thinking about and working on this problem. Neither of us has been satisfied to place foreign substances in the orbit to compensate for the depressed fracture of the floor of the orbit when enophthalmos and diplopia are present.

Nothing can be as satisfactory as replacing the floor and/or the rim of the orbit in its normal, or essentially normal, position. In this way, all the adjacent structures are returned to the positions which they originally occupied and the continuity between them is maintained.

Another factor which must also be considered is that the preferred method should be one which does not require surgical invasion of the orbit.

Our method, which was first presented in the Instruction Program of the Academy in 1951, consists of an intranasal antrotomy through the inferior meatus. The opening is made as small as possible to admit a special pressure balloon which we have devised. After the balloon is placed in the maxillary sinus, it is filled with water under pressure to reduce the fragments. The balloon is then left in the antrum as a splint for ten to fourteen days. At that time, the water is released and the balloon is removed.

One other thing can be said about the permanent antral opening. We firmly believe that any antrum that has had the sinus membrane subjected to trauma is susceptible to future infection, and the antrostomy provides a safeguard to the patient. This water-pressure balloon may not reduce all the fragments at the time of operation, but they are usually back

in proper position within twenty-four hours. By the same principle, the orthopedic surgeon attains similar results by placing a small weight on a fractured leg or arm to reduce the fracture when muscle spasm is great. Our operating time for the balloon procedure is from twenty minutes to one hour. It is very simply performed under local anesthesia and sedation.

We have an additional procedure which is only occasionally required when extreme crushing has been a part of the fracture, causing an enormous number of bone fragments. In such cases, the Anthony-Fisher stainless steel tube jack can be inserted through an opening in the canine fossa and can be left in permanently to hold up the floor and rim of the orbit, after reducing the fracture manually. We also use this tube jack when orbital fractures have healed out of position and require an open reduction, with refracture of the floor, and sometimes of the rim, of the orbit.

IDIOPATHIC THROMBOPHLEBITIS OF ORBITAL VEINS SIMULATING PRIMARY TUMOR OF ORBIT

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BY INVITATION
and
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CLINICAL HISTORY

A WHITE woman, 69 years old, in obvious pain and distress because of a proptosed right eye, reported ocular pain of about two years' duration. Because of the low intelligence of the patient and her family, a satisfactory history was not obtainable. A neighboring ophthalmologist, who had seen the patient at the onset of her symptoms, noted chemosis of the lower conjunctiva but no proptosis. About one month before exploration of the orbit, the pain became severe.

When the patient was examined, seventeen days before orbital exploration, the right eye was proptosed straight forward without other displacement. It could not be pressed back into the orbit. There was marked congestion, but no bruit. Ocular movements were limited, but there was no actual paralysis of the extraocular muscles. No varicosities were present in the eyelids. The pupil reacted to light. Vision was 20/200, corrected to 20/60. Ophthalmoscopic examination revealed slight lenticular haze. The appearance of the fundus was consistent with the age and blood pressure of the patient. Blood pressure was 220/100, but there were no signs of congestive heart failure. The patient was in great pain, but there was neither fever nor evidence of infection. Examination of the nose, throat, and paranasal sinuses revealed no disease. Roentgenograms of the paranasal sinuses gave no evidence of abnormality, and hematologic examinations yielded results within normal limits. There was no history of allergy, hypersensitivity, or parasitic disease, and the peripheral blood showed no eosinophilia.

During the seventeen-day period of observation the fundus picture changed remarkably. The retinal veins became enlarged and tortuous to such a degree that the retina appeared wavy. No hemorrhages or exudates were present. Visual acuity was reduced to counting fingers, and the previously normal visual field became contracted concentrically to 30 degrees. The intraocular tension remained within normal limits.

Although the patient remained afebrile during the entire course of the disease, she was given achromycin and hydrocortisone locally and erythrocin and sulfonamides systemically on a trial basis. These medications produced no improvement in her condition. The orbit was then explored. No distinct tumor mass was encountered, nor was there a hard core of inflammatory tissue of the type seen in inflammatory pseudotumors. In consultation with another ophthalmologist it was decided to eviscerate the orbit for the following reasons: (1) the basic

Dr. Zimmerman is from the Armed Forces Institute of Pathology, Washington, D. C., and Dr. Rogers is affiliated with the Guyton Clinic, Oxford, Mississippi.

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lesion might possibly be a malignant neoplasm, (2) the vision was considered lost, due to secondary vascular changes in the retina, and (3) it would relieve the patient of her severe pain and proptosis.

Postoperatively the patient was extremely grateful for relief of the severe pain. Her convalescence has been smooth and uneventful.

PATHOLOGY

The specimen received in the laboratory consisted of many small irregular fragments of orbital tissue, forming an aggregate which measured 6 by 5 by 2 cm. The largest pieces could be identified as muscle, and several large thick-walled blood vessels were thought to be arteries (fig. 1). Nothing resembling neoplastic or granulomatous tissue was found.

Microscopic examination revealed an inflammatory process involving veins of all sizes. The walls of all these veins were so involved and so thickened that these vessels resembled arteries and arterioles. Elastic tissue stains demonstrated that only the veins were significantly affected and that the minimal arteriolar changes were secondary (figs. 1-3). The process was in different stages of evolution in different vessels. In some, there was acute necrotizing inflammation affecting the entire thickness and circum-

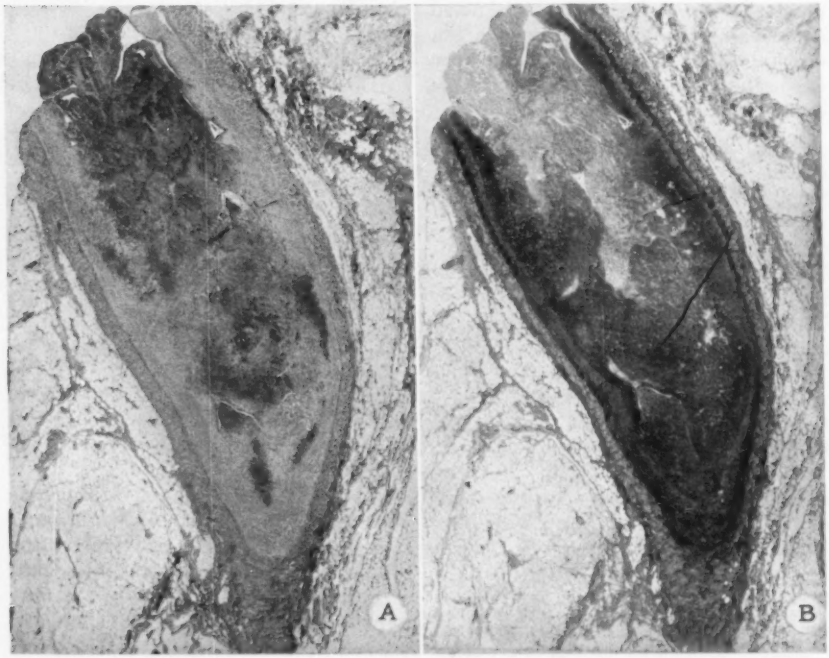

FIG. 1—Because of the gross appearance of the largest vessels, they were thought to be arteries, but microscopic examination proved them to be thrombosed veins. (A) In the hematoxylin-eosin-stained sections, the greatly hypertrophied intima and media mimic those of sclerotic arteries. (AFIP Neg. No. 56-4814.) (B) Stained elastic tissue from same vessel demonstrates conclusively that the vessel is a vein. This vein measured 3 mm. in diameter and is here magnified 13 powers. (AFIP Neg. No. 56-4812.)

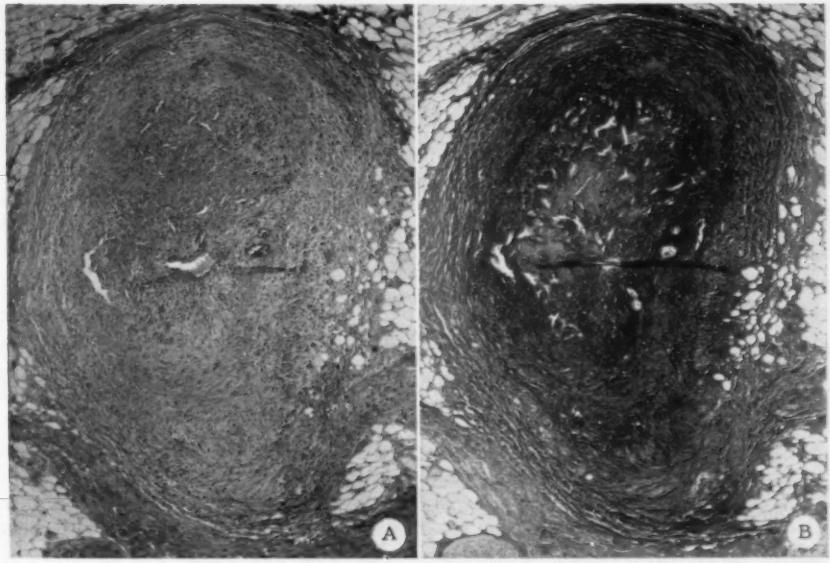

FIG. 2—A smaller vein, which measured approximately 2 mm. in diameter, is completely occluded by a recanalized thrombus. (A) Hematoxylin and eosin. (B) Verhoeff-Van Gieson stain. (AFIP Neg. Nos. 56-4813 and 56-4811, respectively; both ×45.)

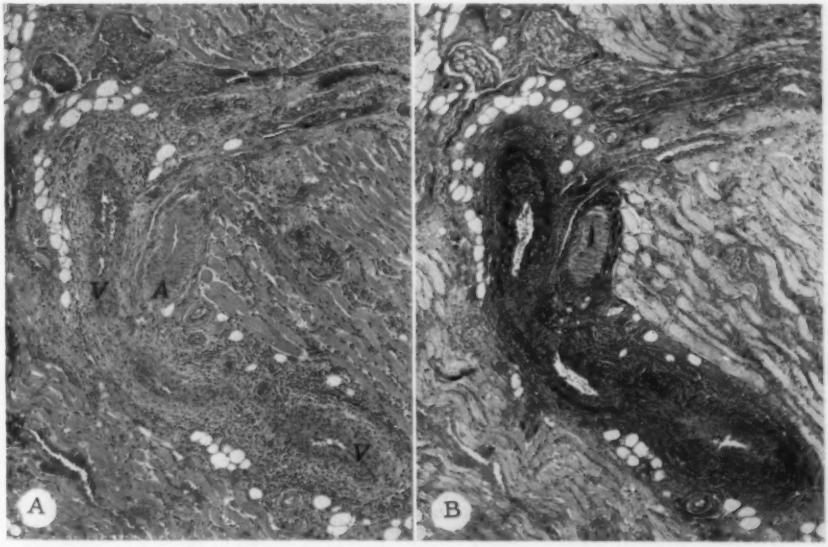

FIG. 3—The small venule (V) exhibits extensive inflammation of all coats, but the accompanying arteriole (A) is uninvolved. (A) Hematoxylin and eosin. (B) Gomori elastic-tissue stain. (AFIP Neg. Nos. 56-4810 and 56-4809; both ×45.)

ference of the vessel wall. In other vessels, only segmental changes were seen. In still others, late stages of reactive hyperplasia with great intimal, medial, and adventitial thickening were observed. As a consequence, the lumens of many vessels, both large and small, were either greatly reduced or completely occluded. The largest vein in the sections measured 3 mm. in thickness, and its lumen was filled with an organizing thrombus which contained areas of recanalization (fig. 1). Marked muscular hypertrophy was noted in its wall.

As a result of these venous changes there was marked exudation of edematous fluid within the orbital muscles. Infiltration by acute and chronic inflammatory cells, including eosinophils, was pronounced. There was neovascularization and an intense reaction of the connective tissue elements within the muscle (fig. 4). Many huge and bizarre mesenchymal cells were seen, but these were quite clearly reactive and not neoplastic.

Due to an unfortunate misunderstanding, the globe was discarded and only the orbital tissue was submitted to the laboratory for pathologic study.

DIAGNOSIS

Our diagnosis is acute and chronic thrombophlebitis of orbital veins, cause undetermined. In view of the fundus changes and visual loss, it seems quite probable that the process had extended to involve the central retinal vein.

DISCUSSION

We have been intrigued by this case because of its puzzling clinical aspects, unusual pathologic features, and com-

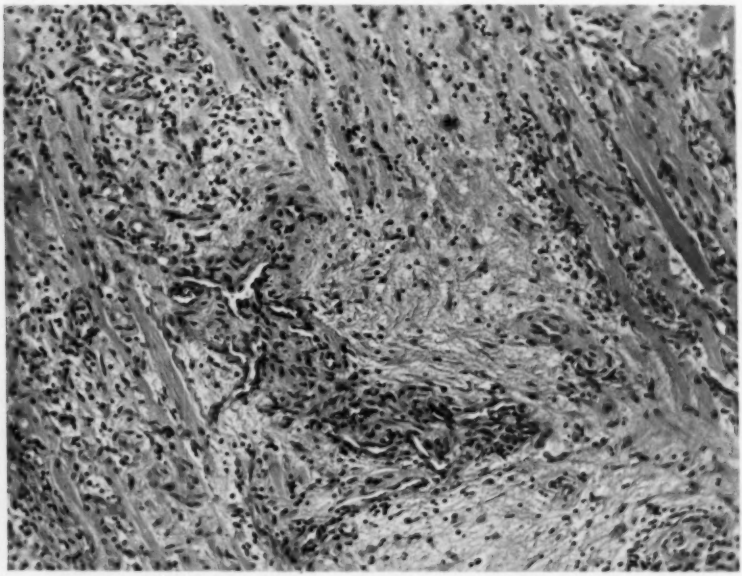

FIG. 4—A section of extraocular muscle reveals a tremendous increase in bulk of the interstitial tissue as a result of increased production of ground substance, retention of fluid, infiltration by inflammatory cells, and fibrovascular proliferation. Hematoxylin and eosin; ×125. (AFIP Neg. No. 56-4806.)

pletely obscure pathogenesis. Thrombophlebitis of orbital veins, once a not uncommon condition, is characteristically an acute fulminating infection associated with great toxicity, severe sepsis, and such grave complications as orbital cellulitis, cavernous sinus thrombosis, meningitis, and pyemia. Most cases develop as a complication of infection of the face, lids, nasal fossae, zygomatic fossa, teeth, or throat, which spreads back into the orbit; or as a consequence of cavernous sinus infection extending forward.[1] Less common causes include penetrating wounds and hematogenous spread from distant sites of suppuration. In the case we are reporting there was nothing in the past history, physical examination, hospital course, or follow-up that would indicate an infection as the cause of the very severe and extensive acute and chronic thrombophlebitis.

Acute exophthalmos has been described as a complication of bland cavernous sinus thrombosis in elderly patients with venous stasis due to severe cardiovascular disease and heart failure.[2] Although our patient was in the older age group and had hypertension, there was no evidence of congestive heart failure and the proptosis developed insidiously rather than acutely. Duke-Elder describes a primary aseptic thrombosis of the cavernous sinus in chlorotic or debilitated individuals at the extremes of life—in marasmic infants and in cachectic persons suffering from such diseases as typhus, phthisis, and carcinoma.[1] The clinical picture of acute proptosis accompanied by limitation of ocular movement, disturbed vision, palsies of the third, fourth, and sixth cranial nerves, sometimes complete internal as well as external ophthalmoplegia, the frequency of bilateral involvement, and grave prognosis serves to differentiate such cases from our own.

Hemangiomas and venous aneurysms of the orbital veins[3] could be confused clinically with the thrombophlebitis present in our patient, except that such vascular anomalies are much more often encountered in children and young adults. Furthermore, our pathologic studies failed to demonstrate anything suggestive of angioma or aneurysm.

Finally, some cases of so-called inflammatory pseudotumor of the orbit are believed to be secondary to thrombophlebitis of unknown cause. Early in such cases an intense eosinophilia may suggest the possibility of parasitic invasion of the orbit or of a hypersensitive reaction in which the orbital veins are the main reacting elements. Typically, however, the thrombophlebitis induces a diffuse chronic inflammatory reaction with much fibrosis, thus accounting for the brawny induration noted upon exploration of the orbit. In our case, two of the most striking features were the complete lack of any palpable tumor within the orbit and absence of diffuse orbital inflammation even on microscopic examination. Only in the extraocular muscles were there secondary inflammatory changes, and these were much less severe than those seen in the usual orbital granulomas.

SUMMARY

A white woman, 69 years of age, complaining of severe ocular pain and marked proptosis of one eye, was found to have an extensive thrombophlebitis of the orbit. The cause and pathogenesis of this process were undetermined. The case is presented as a most unusual pseudotumor of the orbit.

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PRIMARY PLASTIC REPAIR OF NASAL DEFECTS WITH THE NASAL LABIAL FLAP

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SURGICALLY produced nasal defects of limited size can be primarily repaired by using the nasolabial flap.

The removal of a growth from the nose often requires exposure of cartilage or bone. When exposure is necessary, it is impossible to do a primary skin graft and expect the area to heal properly. Skin will not take on bare cartilage and often does not take on cortical bared bone.

A minimal amount of distortion of features and a good cosmetic functional result can be obtained if the incisions are planned to conform as closely as possible to the normal lines of the skin.

The nasolabial flap in the face is attended with little danger; the length of the flap can be three times longer than the width of its base because of the rich vascular supply in this location.

Lines of incision should be carefully planned prior to cutting the flaps, with every effort made to determine where suture lines of the incisions will fall after the flap is rotated. With this procedure the nasolabial fold always delimits the optimum line of closure. The incisions heal well within the crease produced by the nasolabial fold, and often are barely visible after four to five months. The skin lateral to the nasolabial crease, even in a dark-bearded male, is ordinarily relatively free of hair follicles. Unless a flap is to be very sizeable, the growth of hair on the nose in the area in which the flap is placed is no problem. It is sometimes advisable to sacrifice a greater amount of normal skin around the tumor being removed in order that the outlined flap may be cut to follow this principle.

Occasionally it is necessary to fix the nasal ala into a normal position with a deep chromic catgut suture to the periosteum of the superior maxilla in order that the flap may be sutured into position without distortion of the area. This was necessary in a case in which a carcinoma of the nasal ala was completely excised and a flap rotated into position to cover the area (fig. 1; A, B, C).

Remember that a minimum amount of buried suture is always wise in any surgical treatment in which a cosmetic result is important. The buried suture lessens the chances of infection, serum collection, foreign body reaction, or deep tissue necrosis with embarrassment of the circulation.

Careful attention should be given to hemostasis before the flap is sutured into position. Five to ten minutes of pressure with gauze dipped in warm normal saline solution will stop most of the oozing. Large blood vessels should be tied off with 3-0 plain catgut sutures. Application of topical bovine thrombin is an additional help. The flap should always be trimmed and the surrounding tissue adjusted by undermining in order that the flap can be sutured into position without tension. Too much tension on a flap at

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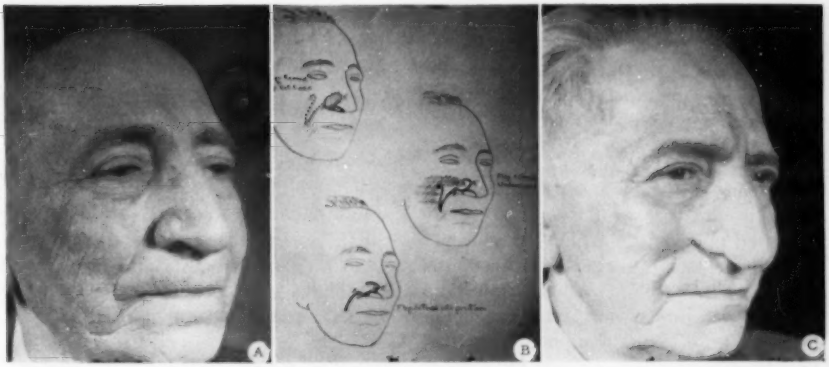


FIGURE 1.

operation is initially indicated by blanching of the skin. If this happens, the sutures should be removed and replaced after additional undermining and adjusting is done to check this condition before the capillaries of the flap are irreparably damaged. Cyanosis at the distal one-fourth or one-half inch of the flap is an indication of poor circulation in this area. It is advisable to trim off this portion of the flap and make the necessary adjustments before suturing the flap in place.

The skin should be sutured with fine cutting needles inserted close to the skin edge to provide careful approximation of the edges of the sutures. I prefer the use of the 5-0 dermalon suture on an atraumatic needle for this purpose, and I usually place my stitches 3 mm. apart and 1 to 2 mm. from the edge of the incision. Postoperatively, a moderate amount of gauze pressure is used over the flap and surrounding undermined area to lessen the possibility of hemorrhage and serum collection and also to enhance any skin approximation. This helps to keep down movement. An elastic type of taping is applied, with the direction of the elasticity perpendicular to the suture lines in order to ensure further release of tension on these areas.

A diet of liquids and soft foods is prescribed for the patient so that there is no unnecessary movement of the tissue in this area. He is given mouth washes and asked not to brush his teeth until healing has been established.

The flap is examined forty-eight hours postoperatively, unless it is necessary to see it earlier because of hemorrhage, undue amount of pain, or fever indicating the possibility of infection.

In the event that a through-and-through excision of the nose is done, it is necessary to bring the rotation flap into position with a skin graft lining. It is preferable to prepare the flap three or four weeks prior to the excision of the area, in order that the skin graft lining the flap may become well established with less chance of fatality to the flap or lining or both.

A ten-month-old youngster had a hemangioma on the nasal glabella area (fig. 2, A). The hemangioma previously had been treated unsuccessfully by a dermatologist using carbon dioxide snow. A complete surgical removal was done (fig. 2, B). Then a high nasolabial flap was elevated (fig. 2, C). This flap was rotated into position and sutured with interrupted 5-0 dermalon. The de-

fect produced by flap rotation closed easily by sliding the widely undermined cheek medially and suturing this to the nasal skin. The result obtained is depicted on this photo taken two years later (fig. 3).

Further use of the nasolabial flap was made in the case of a patient recently operated upon (fig. 4). This patient had an invasive basosquamous cell carcinoma involving the eyelids, orbital tissue and internal canthus region, with extension onto the nose. The tumor board decided that there would be a chance for cure if radical orbital exenteration including contiguous tissues were done (fig. 5, A). A large nasolabial flap was advanced from the right cheek and brought up to cover the exposed infraorbital bone, internal canthus region, and lateral nasal wall (fig. 5, B). A split-thickness skin graft was then used to line the orbit. Split-thickness skin takes amazingly well directly on the bony orbital cavity. The final result was a well-healed nasolabial flap and orbital skin graft (fig. 6, A). The patient is now clinically free of carcinoma. A frosted lens is worn over the exenterated orbit (fig. 6, B).

Complications and the methods of handling them are as follows:

1. Separation at the suture line. The edges of the wound can be reapproximated and held in position with strips of flamed adhesive tape, waterproof type, applied across the edges.

2. Hematoma or serum under the flap or undermined area. A fluctuant area of bulging may indicate either hematoma or serum formation. There may be oozing in one portion of the suture line with hematomas. If the hematoma or serum is at the edge, it is evacuated by gentle expression with mosquito forceps between the slightly opened edges; the edges are then reapproximated with flamed adhesive. If not close to the edge, it can be evacuated through a stab wound with a

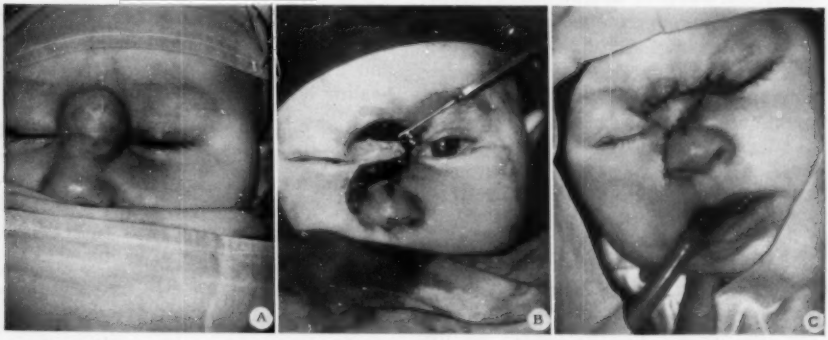
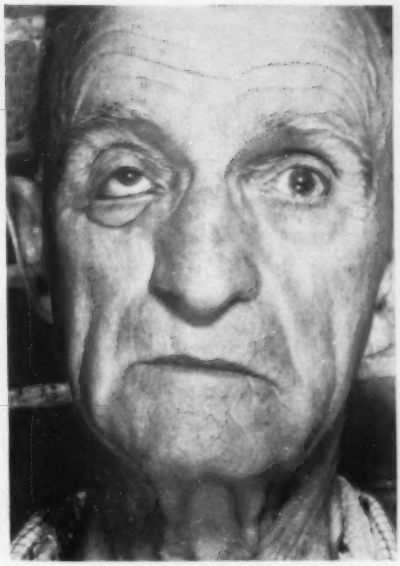


FIGURE 2.

FIGURE 3.



cutting through the skin should be removed whenever possible without losing continuity of the flap. An ice pack applied over the reapplied dressing will help to decrease the swelling and frequently help in obtaining a better cosmetic result when this complication arises.

5. Necrosis at the distal end of the flap. It may only involve the superficial skin of the flap as a demarcated blue or black area. Nothing is to be gained by disturbing this site except for removal of sutures, as separation of the area takes place of itself in ten to fourteen days. At this time the superficial necrotic tissue can be dissected out with small scissors, often leaving the viable deep surfaces of the flap. The deeper layers of the dermis are usually still present and have the appearance of a pinkish mottled dermis closely resembling a skin graft donor site.

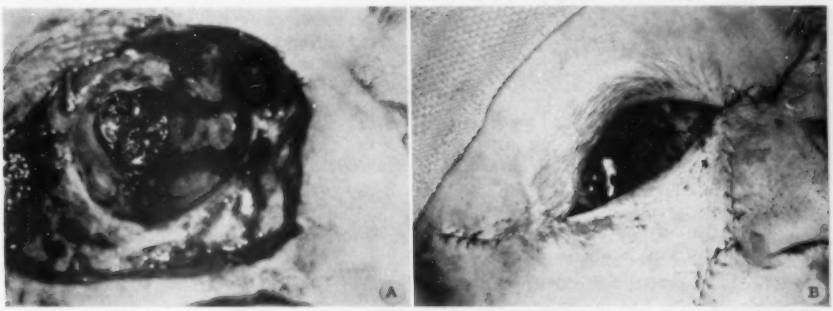


FIGURE 5.

no. 11 Bard-Parker pointed blade. An even pressure dressing is reapplied over the flap.

3. Cyanotic areas at the edge of the flap. Localized cyanosis between sutures may be due to interruption of circulation between the sutures. This may be remedied by removing the offending sutures.

4. Extensive edema of the flap and surrounding tissues. Sutures which are

6. Infection. In the event infection does occur, employ the usual conservative therapy of warm wet compresses for localization and give antibiotics.

CONCLUSIONS

The nasolabial flap is an excellent means of reconstructing nasal defects on the nasal glabellar area, lateral nasal wall, and nasal ala with a minimum of postoperative cosmetic deformity.

FIGURE 6.

DISCUSSION

WILLIAM CLYDE HUFFMAN, M.D., Iowa City, Iowa: Dr. Wynn is to be congratulated on recalling attention to a procedure that seems to be one of the neglected maneuvers in reconstructive surgery. There are many times when such a flap could be used instead of a much more complicated flap or some type of graft. I have never ceased to be amazed at how wide these flaps can be made and still have the donor defect close primarily, particularly in older people. Of course, a flap may be extremely wide and the defect closed primarily even in young children.

The flap may be made two or three times wider than one expects to be possible. The operator intends to close the defect with a graft, but he finds that with extensive undermining, as Dr. Wynn mentioned, it is easy to close the donor area.

CHAIRMAN CONVERSE: As Dr. Huffman said, the amount of tissue that can be obtained from this area is amazing. The case of the child was particularly interesting. In an older individual a lot of tissue is available; in children it is apparently not available, as you have demonstrated.

MEMBER: Does this cause any sensory or motor changes in the upper lip because of that rather large cut?

DR. WYNN: I have never noticed any permanent difficulty with sensation in the upper lip as a result of the flap.

DELMAR FRANKLIN WEAVER, M.D., Detroit, Mich.: Is a graft necessary when the hemangioma is dissected from under the skin? Wouldn't the blueness disappear and the skin there be pretty good without using a graft of any kind? I have had that come up in several cases. I am not citing this case and I am not criticizing it, but I just would like to raise that question and hear what the doctor says about it.

DR. WYNN: I think that is a very good question. Of course in this case that situation would be impossible because the skin here was scarred from previous carbon dioxide therapy. The tumor was extremely adherent to the actual scarred skin in the situation that was presented. You could not peel it out from underneath the skin.

When we are dealing with a subcutaneous hemangioma without skin involvement, we peel the hemangioma out from underneath the skin and preserve the skin; but when the hemangioma actually involves the skin, you have to remove the skin elements also.

PLASTIC SURGERY PRINCIPLES IN HEAD AND NECK TUMOR SURGERY

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THE era of excisional surgery as the only treatment for tumors of the head and neck is passing. The original concept of the management of these neoplasms grew from the frustrated, yet brilliant, efforts of von Langenbeck, Kocher, and others in Europe during the latter part of the nineteenth century. Well-planned technical procedures were complicated by an inability to control the anesthetic and airway systems, severe secondary infection and hemorrhage, and a lack of well-organized data concerning the biology of the tumor. These circumstances were of such serious proportions that hope of surgical progress in head and neck cancer was lost in innumerable tragedies. The bulk of the work was delivered, almost by default, to the roentgenray therapist. After a generation of experience with irradiation, this modality has given way to a new enthusiasm for surgical operation. This enthusiasm, in many instances, is encouraged by the advantages inherent in a direct technical procedure, and superior salvage rates for some types of cancer. Our new era has just begun; its end may be contingent upon the availability of a biologic cure. Surgical intervention in the treatment of cancer has grown to mean more than simply excising a portion of flesh and bone. A conception of composite excision and reconstruction is evolving to keep pace with the efforts to cure and to rehabilitate. A balance between the disease, man's salvageability and his remaining functional self has challenged the surgeon's imagination and courage.

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The application of these principles to surgical operations on tumors of the head and neck is broad. Certainly all plastic surgery techniques and principles are employed in the many unusual situations encountered. Perhaps the most valuable maneuver is the use of the free split-skin graft to cover a wound in the head and neck area either permanently or temporarily. The graft is used with great satisfaction as an immediate dressing in various surgical circumstances relating to the pia of the brain, the dura mater, the galea of the scalp, some areas of cancellous bone of the head, on the face as partial or full replacement, on the neck, in the postmaxillectomy cavity, on the tongue, the palate, the floor of the mouth and the cheek internally, and on the pharynx, larynx, and cervical esophagus. The free split-skin graft has an obvious utilitarian value in extensive surgical procedures of the head and neck.

Free full-thickness skin grafts are valuable in many specific areas of the head and neck where definitive procedures of a smaller scale and of greater refinement are indicated. The supraclavicular, postauricular, and abdominal areas are available as donor sites and are preferred in the order mentioned for color match and texture replacement. The full-thickness skin graft is often indicated in the correction of small defects about the nose, eyelids, cheek, oral cavity, brow, and chin.

Regional pedicle flaps are extremely valuable in dressing and reconstructing wounds of the head and neck area. They are active competitors with free split-

skin grafts and full-thickness skin grafts in many specialized sites in these areas because of their reliability in survival; their sturdiness affords some protection, and often the requirements for hair transplantation, color and texture cannot be supplied by any other technique. They are used extensively in the scalp, on the brow, about the nose, cheeks, and upper and lower lips, and on the tongue, pharynx, lateral neck, and chest. The planning for and use of these regional flaps is part of the continuity of major excisional surgery. The flaps provide a greater chance for a single one-stage operation.

The use of distant pedicle flaps for replacement of tissue in head and neck areas has the obvious disadvantages of protracted morbidity and multiple-stage operations. Fortunately, distant flaps are not frequently required. There are circumstances, however, when there is no alternative except to bring in tissue from a distant area. Tissue from scapula, chest, scalp, upper arm, wrist, and abdomen has been used to advantage in various situations.

Placement of autogenous or homologous bone into a deficient area of mandible as a part of the immediate reconstruction in excisional surgery of the mandibular arch has great advantages over any technique limited to excisional surgery alone. In the past the ascending ramus, head and neck of the mandible were almost routinely discarded in massive operations on the floor of the mouth, the tongue and the neck even though cancer was not present in that area of the mandible. The only prohibitive factors in the immediate reconstruction of the mandibular arch by bone replacement are extreme paucity of intraoral tissue (thus precluding adequate coverage of the bone graft), local infection, and an intensively postirradiated local tissue bed. Bone grafting is a technique to be used later in the frontal area around the orbit and maxilla and has gratifying esthetic results. However, the postradical maxillary cavity is adequately rehabilitated, in most instances, by a dental prosthesis.

An immediate nerve graft, using the autogenous great auricular nerve, is helpful in restoring function to the muscles of the face after a radical surgical operation on the parotid gland in which it has been necessary to sever the facial nerve. A combination of nerve grafting and the other muscular and fascial maneuvers relating to rehabilitation of the face have decreased the ill effects of facial paralysis. Nerve grafting should also be considered in relationship to the ninth, tenth, and eleventh cranial nerves when the nerve pathways have been resected with the operative specimen.

Fat, fascia, and dermal grafts have limited advantages as replacement for soft tissue in areas about the brow, cheek, nose, neck, and chin.

Autogenous fascia lata grafts assist in supporting paralyzed areas of the face, but use of such grafts is probably the least valuable way to rehabilitate the face. Nerve grafting, muscle shortening and tranquilization are all superior. Autogenous fascia is preferred over bovine fascia.

Replacement of blood vessels has given some security in maintaining blood flow to the brain and preventing cerebral hypoxia. The application of general vascular surgical principles to operations on the carotid artery is well established. Fortunately the indications for operations on the carotid artery are rare, yet their accomplishment in some instances is vital. The substitution of an adequate autogenous vein, such as the superficial femoral, or a dry frozen homologous artery implanted in a clean field and covered with a nourishing tissue bed usually succeeds in maintaining continuity of the carotid artery.

SURGICAL TREATMENT OF MANDIBULAR PROGNATHISM

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A GREAT many operations have been devised to correct mandibular prognathism, and not one of these surgical procedures is faultless. On the contrary, each one of them has certain disadvantages as well as advantages.

Operations for protrusion of the lower jaw can be divided into two categories: those performed on the rami and those performed on the body of the mandible. In regard to the former, the commonest technique to be considered is the horizontal osteotomy through each ramus just above the mandibular foramen. Some surgeons do this procedure blindly; others elevate the masseter muscle through a submaxillary skin incision so that a direct view of the ramus can be obtained when making the osteotomy. After repositioning the body of the mandible, some men prefer to wire the fragments together; others do not. In general, this operation gives satisfactory and often very excellent results. However, in some instances, an open bite type of deformity ensues. Naturally, this is an undesirable complication interfering with the mechanism of incising and masticating food. Furthermore, this complication not only is difficult to deal with but also is impossible to correct completely. Some surgeons insist that an open bite will not occur if dental fixation is maintained for a sufficiently long period. However, in my experience, an open bite can occur in spite of prolonged fixation. Another possible, but very rare, complication following osteotomy of the rami is bilateral nonunion. From a functional standpoint, the mandible is completely useless. Repair of such a defect presents an extremely difficult problem.

In the correction of prognathism, some surgeons recommend bilateral condylectomy; others favor osteotomy of the condylar processes. With either technique, the lower jaw can be moved backward. If the condyles are not removed, they can be pinned or wired to the repositioned mandible. Here again, satisfactory results usually are obtained; but an open bite occasionally develops. Not infrequently, a condyle will absorb after pinning or wiring.

Smith and Robinson[4] have recommended the removal of a quadrilateral piece of bone from each sigmoid notch, together with osteotomy of each condylar process. The lower jaw then can be set back in the desired position and the condylar processes wired to the repositioned mandible.

A very complicated operation for protrusion of the lower jaw has been designed by Caldwell and Letterman.[1] It involves osteotomy of the rami in a vertical direction and requires rather large bilateral incisions near the angles of the mandible, reflection of the masseter muscles and periosteum away from the rami, detachment of the internal pterygoid muscles and almost complete osteotomy of the coronoid processes. A vertical osteotomy is then carried down the center of each ramus by means of a dental burr, with care being taken not to injure

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the inferior alveolar nerves. On completion of the vertical osteotomy, the lower jaw is divided into three fragments. The anterior fragment, made up of the body of the mandible and a portion of each ramus, is then telescoped over the posterior fragments until the lower dental arch occupies the desired relationship with the upper jaw.

For many reasons, I prefer not to perform operations on the rami for mandibular prognathism. Some of these procedures are too complicated, some upset the mechanics of the temporomandibular joints, some disturb muscle attachments and some of them can cause an open bite. In rare instances, bilateral nonunion can occur. There is one distinct advantage of these operations on the rami, however; they do not disturb the continuity of the lower dental arch or the body of the mandible.

I favor the older operation of removing a segment of bone from either side of the body of the mandible, which divides the lower jaw into three fragments. When the anterior fragment is brought back into apposition with the two posterior fragments, the deformity is corrected. Needless to say, the disadvantage of this technique involves a necessary shortening of the lower dental arch. However, this disadvantage seems of minor importance when one remembers that this operation does not disturb the joints or muscle attachments and never results in an open bite.

Several years ago, Dr. New and I[3] developed a technique for removing these segments of bone without injuring the inferior alveolar nerves. Some surgeons maintain that cutting the inferior alveolar nerves does no harm because they promptly regenerate. In general, this is true; but I have seen patients who never did regain normal sensation in the lower lip after these nerves have been divided. Consequently, I believe that removal of the segments of bone without injuring the inferior alveolar nerves is well worth while.

The technique of resecting a segment of bone from either side of the body of the mandible can be carried out as a one-stage procedure in which teeth are extracted, the segments of bone surgically removed and the bony fragments immobilized by dental wiring. It is a long, tedious operation that requires submaxillary incisions which communicate with the mouth. Postoperatively, the patient experiences a great deal of swelling, pain and discomfort.

In recent years, I have been doing this operation in three stages, a procedure which has several advantages. The main benefit is a marked reduction in postoperative swelling and discomfort. Furthermore, a lengthy operation is transformed into three relatively simple and short surgical techniques, all of which can be done with the patient under local anesthesia.

Before undertaking this three-stage operation, one should have a dentist secure impressions of the teeth and prepare plaster study models. From these models, the surgeon can determine the width of the segment of bone to be removed on each side. This measurement is represented by the distance from the incisal edge of the lower teeth to the incisal edge of the upper teeth. Furthermore, by means of study models, the surgeon can determine accurately which teeth should be extracted; that is, teeth which are located in areas of bone to be resected. If the patient has all of his lower teeth, I usually extract the lower second bicuspid and first molar teeth which are located in an area of bone best suited for resection. However, if the patient does not have one or more of his molar or bicuspid teeth, the exact position of the segment of bone to be removed can be varied so that more teeth need not be removed.

The first phase of the three-stage operation involves the extraction of teeth that are located in areas of bone to be resected and the preparation of saw cuts through the alveolar process on each side as advocated by Dingman[2] (fig. 1). After extraction of the necessary teeth, the soft tissues on the buccal and lingual side of the mandible in the areas where bone is to be resected are reflected away from the bone. Then, by means of a circular motor-driven saw, the surgeon makes two saw cuts down through the alveolar process, but stays a safe distance away from the mandibular canal. The interval between these saw cuts represents the width of the segment of bone to be removed. At this stage, the bone between these saw cuts should not be removed. Otherwise, the resultant space would fill in with scar tissue which later would prevent proper approximation of the fragments.

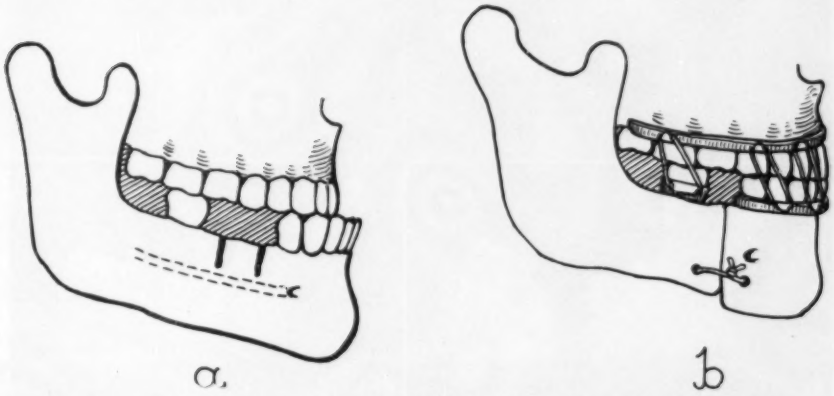

FIG. 1—(a) First stage of bilateral ostectomy for correction of mandibular prognathism. Teeth in each segment of bone to be removed are extracted, and two cuts have been made with circular motor-driven saw from alveolar border, downward, toward mandibular canal. (b) Second and third stages of bilateral ostectomy have been performed. Dental fixation appliances are in place. Segments of mandible have been removed. Fragments held together with heavy chromic catgut if teeth are present in lower fragments for fixation. If no tooth is present in posterior fragment, tantalum wire, rather than catgut, holds fragments in apposition. (From Erich, J. B.: Treatment of prognathism of the mandible, The Journal-Lancet, 76:247-253 [Sept.] 1956.)

Subsequent to extraction of the teeth and preparation of the saw cuts through the alveolar process on each side, the intra-oral wounds are sutured and allowed to heal for from three to four weeks. This first stage can be performed with the patient under general or, as I usually prefer, under local anesthesia. After three or four weeks, when the intra-oral wounds are healed, the second and third stages of this operative technique can be carried out. The second stage involves the removal of a segment of bone from one side of the mandible; the third stage, the removal of a segment of bone from the opposite side.

Before proceeding with the second stage, dental appliances for fixation of the teeth should be attached. In my opinion, hooked arch bars and elastic traction should be used for this purpose. Elastic bands not only bring the teeth into the best possible state of occlusion but also effectively immobilize the fragments of the lower jaw. In the two lower posterior fragments, only one or two molar teeth, if any at all, are present. It is difficult to

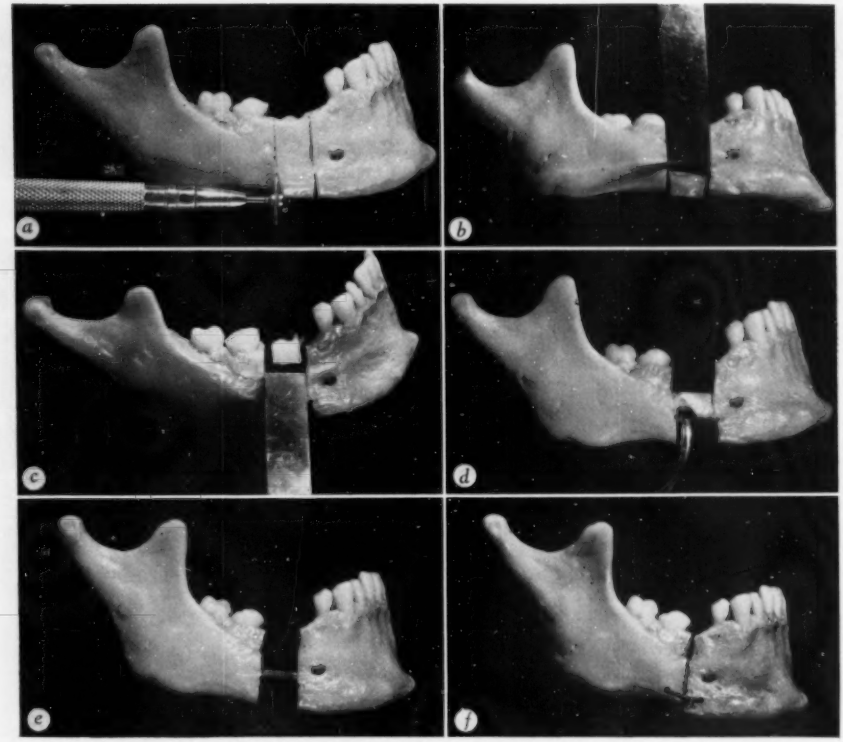

FIG. 2—Removal of segment of bone, without injuring inferior alveolar nerve, in cases of mandibular prognathism. (a) Teeth in segment of bone to be removed have been extracted one month previously and saw cuts have been made with circular motor-driven saw from alveolar edge, downward, close to mandibular canal. Circular saw makes two cuts from the lower border of the jaw, upward, close to mandibular canal. (b) A chisel removes intervening bone between the lower saw cuts. (c) Chisel removes bone intervening between two saw cuts made one month previously. (d) Careful use of rongeurs removes remaining bone around inferior alveolar nerve. (e) Segment of bone completely removed without injuring nerve. (f) After segments have been removed from each side of body of mandible, the anterior fragment is moved back into apposition with posterior fragments and they are held together with catgut or tantalum wire. (a, b, d and e are reprinted from New, G. B. and Erich, J. B.)[4]

wire a single molar tooth satisfactorily. Under these circumstances, I prefer to use an orthodontia anchor clamp band around the tooth rather than a wire. When screwed down around the tooth, such a band cannot be dislodged, and its buckle sheath makes a good attachment for rubber bands.

The second stage of this operation can be carried out to advantage with the patient under local anesthesia. I prefer to block the inferior alveolar nerve with a 2 per cent solution of piperocaine hydrochloride (Metycaine®) after which a 1.5 per cent solution of Metycaine is infiltrated around the body of the mandible at the operative site. If the patient is apprehensive, sufficient thiopental sodium (Pentothal Sodium®) is administered intravenously to produce analgesia. With this minimal amount of solution, the patient is not asleep but is no longer concerned about the surgical procedure.

A skin incision 2 in. long is made 2 cm. below the free border of the man-

FIG. 3—Marked mandibular protrusion corrected by three-stage bilateral ostectomy of the body of the mandible. Nasal plastic operation was performed to correct nasal deformity. (a) Preoperative lateral view. (b) Postoperative lateral view. (From Erich, J. B.: Treatment of prognathism of the mandible, The Journal-Lancet, 76:247-253 [Sept.] 1956.)

FIG. 4—Marked mandibular prognathism corrected by three-stage bilateral ostectomy of the body of the mandible. (a) Preoperative lateral view. (b) Postoperative lateral view.

dible. This incision should be made low enough to miss the twig of the facial nerve innervating the lower lip. By blunt dissection, the soft tissues are pushed aside from the body of the mandible buccally and lingually. In view of the fact that the teeth in the area of bone to be resected were extracted one month previously, the wound prepared in the submaxillary region should not communicate with the oral cavity; this is distinctly advantageous. On retraction of the soft tissues, the two saw cuts that were made in the alveolar process one month earlier can be seen easily through the submaxillary incision. Then with a circular motor-driven saw, two cuts are made from the lower border of the mandible upward, staying a safe distance away from the mandibular canal (fig. 2, a). By placing a chisel between these two cuts, the intervening bone is knocked out (fig. 2, b). In similar fashion the bone located between the saw cuts in the alveolar process is removed (fig. 2, c). Now around the inferior alveolar nerve and vessel only a small amount of bone remains; this can be nibbled away with a small rongeur (fig. 2, d), leaving the nerve intact and uninjured (fig. 2, e).

Next, a hole is drilled about 1 cm. beyond the cut edge of each lower fragment. Heavy chromic catgut or wire is inserted through these holes and the bony fragments are tied together in proper apposition (fig. 2, f). If teeth are present for fixation in a lower fragment, I advise the use of heavy chromic catgut, rather than wire, to approximate the fragments; wire on rare occasions can cause trouble necessitating its removal. If there are no teeth in the posterior fragments, then, of course, the fragments must be tied together with wire; I prefer tantalum wire for this purpose.

The external wound in the soft tissues is now carefully sutured with fine silk, which completes the second stage of this operation.

Two or three days later, the third stage is undertaken. This procedure is merely a repetition of the second stage on the opposite side of the mandible. Whenever possible, I prefer to carry out the third stage with the patient under local anesthesia as previously described. This permits the application of intermaxillary rubber bands bringing the teeth into occlusion and immobilizing the fragments as soon as the operative procedure has been completed. If general anesthesia has been employed, I hesitate to wire the teeth together for several hours until the period of vomiting has passed.

Some surgeons do not approve of dividing this operation into stages. However, when it is performed in stages, the incisions in the submaxillary regions do not communicate with the mouth, thereby reducing the amount of inflammatory reaction in the wounds and, in

FIG. 5.—Roentgenogram of left side of mandible. Fragments held together with tantalum wire, since there was no upper tooth to which the molar tooth in the posterior fragment could be wired for fixation of this fragment.

turn, the degree of postoperative swelling and discomfort for the patient. Also, it permits the wounds to heal in a much shorter time. Finally, I want to emphasize the fact that operating on one side of the mandible at a time diminishes the amount of discomfort which the patient experiences at any one time. In my experience, this three-stage procedure has been more satisfactory for the correction of mandibular prognathism than any other method which I have employed (figs. 3, 4, and 5), and it has caused the patients far less disagreeable postoperative symptoms than have occurred with other techniques.

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DISCUSSION

V. H. KAZANJIAN, M.D., Boston: The purpose of surgical treatment of mandibular prognathism is to improve the contour of the lower portion of the face and give better functioning power to the teeth. There is, however, considerable difference of opinion regarding the type of operative procedure to be used. As Dr. Erich has pointed out, one method consists of removing a measured section of bone from each side of the body of the mandible, and then pushing the anterior segment back to a new position, where it is immobilized. Dr. Erich does not approve of this second procedure; this is where we disagree. Mandibular protrusion is not a uniform pattern in all patients. For example, some cases show a contracted upper dental arch combined with an abnormally large mandible, while others are complicated by an open bite in addition to the prognathism. In some cases the patient may have a number of missing teeth. With these facts in mind, it is evident that one type of operation is not suitable for all conditions. The relative merits of these two operative procedures depend upon the type of deformity and the condition of the existing teeth.

There are disadvantages, as well as advantages, for each general type of operation. The removal of a measured section of the body of the mandible (ostectomy) necessarily exposes the operative field to the oral cavity, and the hazard of infection must be considered, although this is probably a minor hazard at the present time. Careful surgical technique, combined with the modern means of preventing infection, should minimize the chance of infection. A second hazard is the chance of injury to the mandibular nerve, which causes anesthesia of the lower lip. In most cases the normal sensations gradually return, but there are some cases in which the anesthesia has been permanent. However, the most important objection to ostectomy through the body of the mandible is that although this procedure fulfills one objective of the operation—improvement of the general appearance of the face, it does not always attain the other objective—maximum efficiency of the masticatory function of the teeth. In order to remove a section of bone from the body of the mandible, at least one or more of the lower teeth will have to be removed from each side. Also the upper third molars will be useless, since they will not be in contact with the lower teeth. If the entire mandibular arch is pushed back by sectioning the ascending ramus, one also acquires, in most cases, maximum efficiency of the masticatory power of the teeth. Osteotomy through the rami, above the level of the mandibular canals, is probably a more simple operative procedure, and special splints are not required for immobilization of the lower jaw. There is no interference with the inferior alveolar nerve, and greater efficiency in mastication is obtained because no teeth are sacrificed. I think that this is the most important factor in favor of the procedure. However, in order to be efficient in this type of procedure, one must

select cases that are suitable for osteotomy through the ramus. I have found that patients with mandibular protrusion combined with open bite, or edentulous or semi-edentulous cases, should be operated upon by ostectomy through the body of the mandible.

SIDNEY K. WYNN, M.D., Milwaukee: I would like to defend the bilateral sectioning of the mandible below the coronoid notch. I don't recall ever having had one operation result in an open bite when the case had been studied preoperatively with a dental model. However, I do sectioning of the body of the mandible in selected cases.

I enjoyed Dr. Erich's paper very much because I think his results are really par excellence.

I believe the results of every method used depend upon the operator, and that all methods have advantages.

DR. ERICH: I want to thank the discussers for their comments on this problem of mandibular protrusion. I did not intend to give the impression that I am condemning operations on the rami. Instead, I merely wanted to point out their disadvantages, and to emphasize my reasons for preferring ostectomy of the body of the mandible.

IMMEDIATE TREATMENT OF LID LACERATIONS

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DETROIT, MICHIGAN

TRAUMA and eye injuries, especially lid injuries, are now caused more frequently by home and automobile accidents than by industrial accidents. Most of the injuries I treat result from accidents caused by such instruments as a .22 revolver, an ice pick or a knife. These accidents are more common than is realized. Most of them occur at night.

Immediate treatment will decrease the hospital stay of the patient and will enable the physician to use tissue that soon becomes necrotic due to the lack of blood supply. Early repair is imperative, since secondary edema occurs minutes after injuries to the orbit.

First and foremost in the repair of lid injuries is careful inspection of the parts. Make sure that there are no depressed fractures that will require further repair later. The physician must take time to do the best job possible even under emergency conditions.

The injury which results from a tear presents a different problem than injuries caused by glass or a knife. The physician must repair any deep injury first, carefully approximating the deep tissues, since the scar will extend from there up to the surface. Remember the contraction bands come from the deep tissue, not the superficial.

Occasionally I must treat an injury of passion in which an eyebrow is bitten and avulsed. A new eyebrow must be supplied immediately. A skin graft from the back of the neck may be used.

If the lid is badly torn from the nasal side, the surgeon must realign the tissue before he sutures. The internal canthal ligaments must be attached to the periosteum of the nose. In some cases, the lacrimal canals must be reconstructed. This is a time consuming operation. The deep tissues must be sewn first, using the minimum amount of catgut, causing no more trauma than necessary, and keeping the under surface of the lid as smooth as possible.

Examine even small, horizontal linear lacerations carefully. In one instance the levator muscle had been torn from the superior margin of the tarsal cartilage. The repair can be done before the lid laceration is sewn. If the tearing is unnoticed or neglected, a ptosis operation will be necessary later.

If the surgeon waits until the results of the first operation are final and the lid is healed, he may find secondary repairs unnecessary.

One patient was treated for a lid wound resulting from a dog bite. The dog's tooth had entered the temporal side of the lid and tore down sharply, cutting it through. In such cases, the under surface of the lid should be sewn together immediately, but, most important, be sure that the levator has not been cut through and that the injury does not extend to the side of the eyeball, resulting in severed extraocular muscles.

Some lid injuries are very extensive. They may involve the eyeball or both sides of the nose. If the nose is broken, the surgeon may have to build up the inferior plate of the orbit. Sometimes, he may have to wire the zygomatic process

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A condensation of a commentary on slides.

Presented at the Sixty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 14-19, 1956, Chicago.

into position before he can sew the lids.

Injuries along the nose which result in a torn punctum are serious since the lower lid is important in keeping the cornea moist. The gutter of the lower lid, running from the lacrimal gland, touches the lower margin of the lid. If the lower lid falls away, the lower third of the cornea will dry out. Desquamation and a secondary infection may follow.

Immediate repair of the lid should be just that—immediate. Take all the time that is necessary to repair it.

TREATMENT OF THE LATE RESULTS OF LID LACERATIONS

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WITH proper and adequate repair of acute simple lacerations of the eyelids, most deformities are avoided or are of only minor significance. When only insufficient or emergency measures are carried out, the acquired abnormalities of the eyelids following trauma can be many and varied, indeed often bizarre. Ectropion, entropion, ptosis, epicanthus, misalignment, partial or complete loss of eyelids, and other abnormalities are found singly and in combination. Success in ophthalmic reconstructive surgery depends largely upon the interpretation of the deformity presented and the ability to restore proper anatomic relationship and physiologic function.

Just how long one should wait before initiating secondary repair poses a question. Certainly, immediate steps must be made to restore the physiologic protection of the cornea. Roentgenograms should be made to find possible retained foreign bodies and fractures or displacements. When it is evident that cicatricial contraction is producing ectropion or entropion of minor degree, a splinting of the margins with lid adhesions for several months may correct that deformity. Keloid formation is treated by low voltage roentgen therapy before and after excision. Some forms of paresis, firm edema, lagophthalmos, and odd-appearing scars slowly resolve into well-functioning and pleasing contours. In general, allow full healing to take place so that the full final effect is known. Ordinarily, at least three months are required for scar tissue to contract and become relatively avascular; it takes much longer in severe cases. Do not operate, as a rule, before the healing process is completed. Deep cicatricial tissue can be softened and suppled with deep massage, which should be started six weeks after the operation.

Delayed unfavorable effects of lid lacerations are caused by contracting scar tissue and disruption of the supporting structures, such as the tarsi and canthal ligaments, together with unopposed pull of muscle tonus.

Simple notching of the lid margin calls for excision of the tense scar band and re-creation of the original wound. The choice of repair for simple wounds is the time-honored halving method of Wheeler.[13] The tarsoconjunctival layer is excised on one side and the skin and orbicularis on the other. Multiple wounds do not allow the extravagance of normal tissue excision. The tongue-and-groove method of Hughes[5] does not waste tissue; a groove is made at the gray line on one side of the wound and a tongue wedge of tarsus is made on the other side. These are fastened together with a through-and-through double-armed suture near the lid margin. A flap of skin adds support. Another choice is the Minsky[8] "figure of eight" suture through the gray line, giving exact alignment and using the opposing lid for support. For opposing lid wounds the "double figure of eight" works well. Occasionally, the best method is to advance a tongue of the tarsoconjunctival layer from the opposing lid.[3]

A notch in the nasal end of the lower eyelid allows the temporal portion of the

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lid to be displaced downward and outward. Overcorrection, with the denuded tarsal end tucked under the attachment of the medial canthal ligament and held with a double-armed silk suture, emerging well beyond, will allow a more normal position when healed. Subcutaneous sutures in layers hold the conjunctiva and orbicularis fibers together and relieve undue stress on the tarsal tongue. Sometimes when re-creating the original wound and excising scar tissue, it is possible to locate the medial end of the severed canaliculus. With a needle, nonabsorbent suture, such as dermal, may be threaded through the punctum into the sac, where it emerges through the apex and is tied in a loop. In some cases it may be considered worth while to attempt plastic construction of a new entrance into the sac by a conjunctivodacryocystostomy by the method recently reported by Jones.[7] Jones also gives an excellent description of the detailed anatomy of the medial canthal region.

When wounds in this region are more extensive, the lacrimal sac is usually sacrificed and excised. Displaced bone is removed. The lower lid is mobilized by lateral canthotomy and by severing its inferior, posterior attachment. The posterior limb of the medial canthal ligament is fastened to the posterior lacrimal crest by heavy chromic catgut or wire.[1,7] The anterior limb is sutured in overcorrection as previously described. Additional support can be gained by the interpositioning of a flap of skin from the nasal portion of the upper eyelid. This is placed below the elevated canthus.[10] Disruption of the medial canthal ligament and traumatic epicanthus are treated by fastening the complete ligament in an elevated position with wire through the superior portion of the posterior lacrimal crest. A crescent of excess skin and subcutaneous scar tissue is excised and closure made with buried catgut attached to the periosteum of the anterior lacrimal crest. Silk is used for skin closure.

Intimate knowledge of the regional anatomy is necessary in order to treat displacement of the outer canthus.[4] The approximation of the lid to the globe depends upon the attachment of the posterior limb of the canthal ligament to the periosteum in the region of the lateral orbital tubercle. When possible, attachment is best accomplished with wire through small holes drilled in the lateral orbital wall 3 mm. back of the anterior margin. The orbicularis fibers are attached to the periosteum anteriorly in a crisscross fashion to give proper pull and support to the lids. This helps make the acute angle characteristic of the lateral canthus. Raising or lowering of the angle can be made by interpositioning *Z* of skin flaps after the ligament is put into proper alignment.

Entropion due to scarring and shortening of the tarsoconjunctival layer results in irritation to the globe from inturning cilia. The buckled upper tarsus is everted by the modified Ewing[12] procedure. A split is made through the tarsus from the inside, 3 mm. above and parallel to the lid border. Four double-armed sutures are placed horizontally through the superior edge from within and emerge above the lash margin in a vertical fashion. The ends are tied over a small catheter to produce an outward buckle in the healed position. Entropion due to symblepharon and shallow cul-de-sac call for a conjunctivoplasty or mucous membrane graft. Sutures or conformers must be used to hold the newly formed fornices until healed.

In absence of the globe, the socket frequently needs to be rebuilt. The method of reconstruction of the bony walls by bone, cartilage or tantalum has been presented by Callahan.[2] Volume can be built up with deep acrylic implants and

wedges along the floor.[11] This helps to eliminate the deep sulcus beneath the superior orbital margin. The socket should be lined with mucous membrane grafts when possible. Avoid a combination of skin and mucous membrane.

Ectropion produced by cicatricial contracture not relieved by the conservative measures mentioned above requires lengthening of the vertically shortened cutaneous surface. Excision of the scar bands and application of a suitable graft are usually necessary. A thin split-skin graft may be used to supply additional skin. Preferably, however, use a full-thickness graft from the upper eyelids or retroauricular areas, when possible, for replacing the palpebral skin. Use of tarsorrhaphy is advised during the healing phase of about three months. If the ectropion has persisted, the lid border may be stretched. A wedge of tarsus can be removed near the lateral canthus in order to shorten the lid. A lateral tarsorrhaphy, which does not include excision of the eyelashes of the upper lid, will improve a wide palpebral fissure and help elevate the lower eyelid. If only the lower lid is involved, a double-pedicle skin graft from the upper eyelid is swung down in hammock fashion for tissue replacement and support. All grafts are sutured in place with interrupted 6-0 silk.

In treating lacerations involving the upper lid, a complete or partial blepharoptosis often accompanies lid lacerations. I wish to emphasize that it is possible to repair this blepharoptosis as a secondary procedure. Circumstances may have prevented the repair at the time of the injury. The ptosis may be due to severance of the levator tendon from some but not all of its attachments. It may be due to a sharp laceration across a major portion of the tendon. Ordinarily only the portion attached to the tarsus, orbicularis, and skin is missing. The lid sags; the tendon retracts but is held laterally by the attachments to the orbital tubercle and medially to the posterior lacrimal crest and trochlea. An external approach is made through the upper eyelid along the line of the lid fold some 6 to 8 mm. above the lid margin. The upper border of the tarsus is identified. Search for the severed tendon behind the orbital septum. Tracing back from the lateral or medial attachment is definitely helpful. Local anesthesia and the patient's voluntary control of the levator also help in the identification. Once found, the severed tendon may be brought forward to the anterior surface of the tarsus. Some of the tendon may be resected depending upon the degree of ptosis. A new lid fold is assured by biting into the advanced tendon or tarsus with the silk sutures that close the original skin-orbicularis incision.

When there is complete loss of eyelid substance, including the lid margin, replacement of both the tarsoconjunctival layer and skin-orbicularis layer is necessary. For a loss involving one third or less, a sliding advancement flap of the Celsus (as modified by Knapp, Wheeler, and others) type will serve. This can be applied to either upper or lower eyelid. A thorough lateral canthotomy, including the posterior attachment of the lateral canthal ligament of the respective lid, is performed for good relaxation. A Burow triangle excision is made at the base of the flap. Deep sutures are placed into the periosteum at the lateral canthus to assist in the pull and to relieve some of the tension at the tip of the flap. The cut edges of the lid are brought into position by the halving procedure.

If the area lost is greater than one third of the lid, either subtotal or full thickness of the eyelid, the Hughes[6] method of reconstruction is ordinarily preferred. The exact indications and detailed techniques are well presented in his book on eyelid reconstruction.

For replacement of eyelashes, a graft 3 mm. wide can be taken from the midportion of the nasal one third of the eyebrow. For the lower eyelid the graft is taken from the contralateral eyebrow and the nasal end of the graft remains near the nose. The grafting should be done while the tarsorrhaphy is still present.

The eyebrow can be replaced by a free graft from the parietal or occipital portion of the scalp. On occasion the opposite eyebrow may be full enough to permit half of it to be transferred. The successful take of these grafts depends upon a base with a good blood supply and free of scar tissue. The graft should be thick enough to protect the root bulbs and should be handled gently with hooks. The margins must be placed in good apposition with fine sutures. Circumstances may dictate the use of the tattoo methods of Beard if the chances of failure are great. This also applies to grafted lashes that are scanty or of poor quality. The excision of individual or group lash roots may be necessary in repairing marginal defects. Do not forget that artificial lashes and eyebrows are possible for those willing to learn the proper trimming and application.

All surgical reconstructive procedures about the eyelids heal better with dressings that do not stick to the tissues. The dressings should provide moderate pressure; they should be applied with a head bandage to control swelling and prevent displacement of the muscles and other tissues.

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TREATMENT OF DEPRESSED AND ADHERENT SCARS

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DEPRESSED and adherent scars have two common etiologic factors: faults in the primary closure of the original injury, regardless of whether the injury involves soft tissue only or tissue and bone, and post-traumatic infection. These factors are the result of (1) failure to make accurate approximation of deep and superficial tissues; (2) loss of soft tissue as a result of injury or secondary infection; (3) failure to reduce orbital fracture before bone fragments become fixed in malposition. The treatment by ophthalmic plastic surgery of scars due to failure or loss will be discussed in this portion of the symposium.

Failure to Make Accurate Approximation of Deep and Superficial Tissues

The first type of case is that in which the wound was closed by primary sutures without an accurate approximation of the deep and superficial tissues. This type of closure frequently occurs when it becomes necessary, following an accident, for a surgeon not acquainted with the detailed anatomy of the lids to perform an emergency operation to close the laceration. Occurring as frequently, and with equally bad results, is primary closure done by young, untrained, surgical residents or by interns even less skilled. These operators feel that it is necessary to effect closure as best they can; they often use heavy sutures which are passed through both the superficial and the deeper tissues, disregarding the necessity for closure of the structures of the lids layer by layer. The resultant scar is probably the most common type seen by the ophthalmic plastic surgeon. No soft tissue has been lost. The defect is due to poorly performed primary repair (fig. 1).

Loss of Soft Tissue

The second type of case, not too much unlike the first, is that in which soft tissue had been actually lost as a result of the injury, or lost because of secondary infection after the primary suturing (figs. 2 and 3). The technique for satisfactory secondary repair differs for the two types of cases.

Failure to Reduce Orbital Fracture

The third type of case is that in which depressed fractures of the rim, and/or floor of the orbit, or of the zygoma has resulted in a depressed scar. The excuse made all too frequently by the surgeon or ophthalmologist who first treated the case is that he believed the patient to be so seriously injured that primary reduction of the orbital fracture was contraindicated. Such a statement shows poor judgment. Many of these cases will need several days of treatment for combatting the primary shock, but certainly the largest number of them could be reduced surgically before the bone fragments have become fixed in their malposition by early healing (fig. 4).

I feel that the discussion of specific cases will best illustrate these essentials.

Excluding the third of these types for the moment, the first two categories must be studied preoperatively, in order that the operator may decide (a) whether

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FIG. 1—(A) Appearance of patient at first visit. Medial lip of wound in ectropion, lateral lip in entropion. Scar adherent to the rim of the orbit, to the malar and to the right nasal cartilage. Deep punctured scar is adherent to a depressed fracture of the maxilla. (B) Postoperative result.

FIG. 2—Depressed, adherent scar with very evident loss of external layers of skin, of the lid, and the inner canthal angle.

FIG. 3—Another view of the deeply adherent scar, and of the lost soft tissues.

soft tissue, deep or superficial, has been lost; (b) if it has not been lost, what plan is best for secondary closure; and (c) if it has been lost, whether tissue for replacement can be obtained, either as a free skin graft, full-thickness or otherwise, and/or a pedicle flap. This can be readily done with preoperative photographs and with sketches illustrating the scars and the tissue defects and visualizing the situation which would be present after the scars have been resected. In these cases, it is most important

FIG. 4—(Left) Depressed, adherent scars, with loss of bone of the zygoma and of the upper outer angle of the roof of the orbit. Traumatic anophthalmos. (Center) After scar resection and cartilage graft. (Right) With prosthesis. (Photophobia is from the light used for photography).

to arrive at some conclusion relative to these aspects and to plan the operative technique on the basis of such conclusions.

Factors connected with the actual operation are exacting as to performance, but the procedure is not especially difficult. Perhaps the first and most important factor is the closure of those areas from which a depressed scar has been resected. All of the scar must be resected, no matter how deeply it may have penetrated or how extensively it may have spread. (At this time the handling of keloids is not being considered.)

The closure of a resected defect must be in anatomic layers (fig. 5). After the scar resection, the conjunctiva and the orbicularis fibers in the lid need separate closure with catgut. The deep fascia on the face must be sutured separately in a suture line, also with catgut, and offset from the deeper and the more superficial lines of suturing. The last step is an accurate approximation of the skin edges with silk sutures.

Two basic rules are important in these closures. Tissues must be supported as they are elevated, and long linear scars should be broken up to eliminate the pull of subsequent cicatricial contractures (fig. 6).

Figure 7 illustrates closure of the wound seen in figure 1. The preoperative sketch to the left shows that there is no loss of soft tissue. The problem, therefore, is simply one of scar tissue resection and proper secondary closure.

The medial lip of the cicatrix is in ectropion, but the lateral lip is in entropion. The cicatrix itself is adherent to the rim of the orbit for two thirds of its length. The scar is also adherent to nasal cartilages. After all scar tissue has been adequately removed, the re-establishment of a linear cicatrix is prevented by swinging the major flap, on the lateral side of the resected area, into one of the gaps on the medial side; in this way, those tissues which re-formed the lower lid and the conjunctival cul-de-sac and corrected the lid margin deformity are supported.

The closure of the deeper layers is important. If the orbicularis fibers, including the fascia of the lid, are properly joined, the skin flaps will usually fall into place (fig. 8).

Patients will occasionally have depressed scars, wherein it is necessary to replace subepithelial tissues which have been lost. We shall not include at this moment the consideration of bone defects. In some cases a two-stage operation is necessary: (1) a resection of scar, with secondary suturing; (2) some type of implant (fig. 9). In other cases a depressed cicatrix can be resected and the implant made simultaneously (fig.

FIG. 5—Scar resection and resuture in layers. (Left) Defect as first seen. (Right) Surgical end result. The right eye is a prosthesis. The eye was lost at the time of injury.

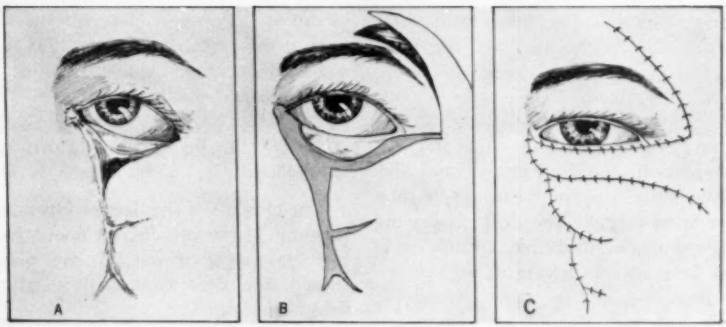

FIG. 6—Original sketches for correction of a case, illustrating certain basic principles. (A) Laceration caused by a broken windshield. The wound had not healed well following wretched primary suturing. It is quite evident, when studying this case, that soft tissue has been lost. (B) The situation after the scars have, theoretically, been resected. The conjunctiva, still intact, is spared; a pedicle flap from above is outlined for the correction of the ectropion, and for the replacement of the lost tissues. Simultaneously, the elevated tissues are supported in the secondary suturing, and at the same time the perpendicular pull of the healing cicatrix is broken up. In this case it is quite likely that a full-thickness, free skin graft would have been as satisfactory as the pedicle flap. (C) Silk suture lines for the skin in the secondary closure were not introduced until deep 6-0 catgut sutures had completed the closure of the deeper tissues satisfactorily. Skin sutures alone would not be sufficient.

FIG. 7—Sketches of the lid laceration illustrated in figure 1, and of the steps in the technique planned for the correction of the defect. The satisfactory result is illustrated in figure 1, B.

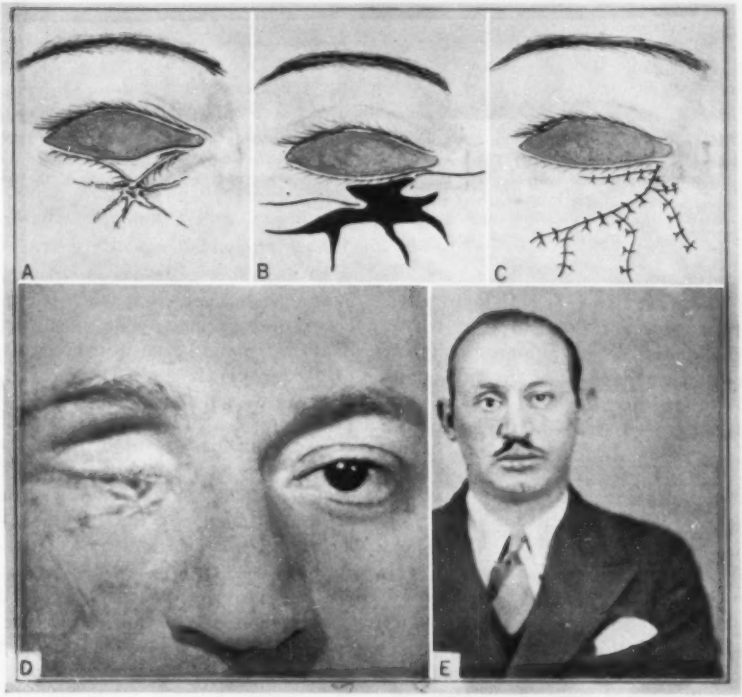

FIG. 8—Composite illustration emphasizing the importance of proper closure of the deeper subepithelial layers. In this case the eyeball was lost at the time of the original injury. (A) Scar to be resected. (B) Resection. The proper closure of the orbicularis fibers, including the fascia of the lid, was the most important part of the operative correction in this case. (C) The skin incision line needed no planning; the skin flaps almost fell into place.

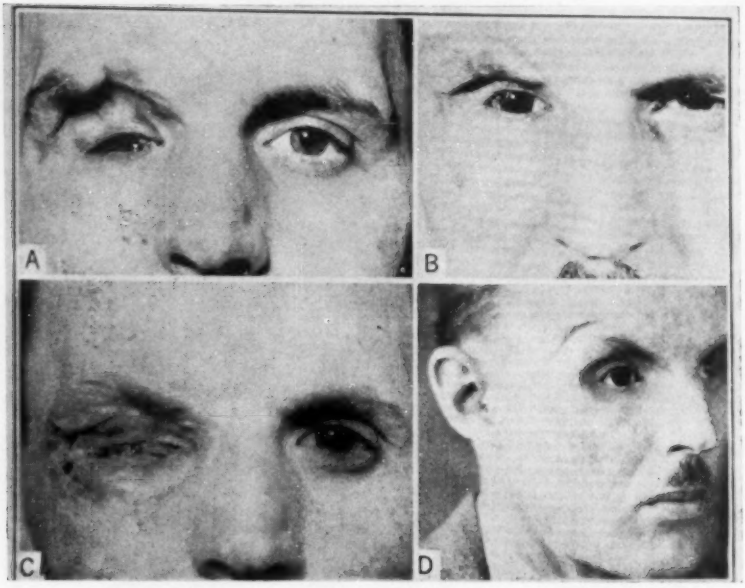

FIG. 9—Sliding pedicle flap with fascia implantation for depressed, adherent scar. (A) The original injury. Right eye is lost. (B) The result following scar resection and a sliding flap to replace the eyebrow in its normal position. (C) Postoperative condition of fascia implant at time of the removal of sutures. (D) Completed end result.

10). There are two very satisfactory tissues available for this purpose: tensor fascia femoris, as an autogenous graft, or cadaver cartilage—or even autogenous cartilage, if the case is sufficiently extensive to need that—in the shape of plaques, shells, discs or wedges.

When utilizing fascia one must be very careful that complete hemostasis has been achieved before the fascia implant is sutured into position. Also, it is necessary that there be a closed layer of subepithelial tissue over the implant. The suture line for this layer should not be parallel to nor in contact with the suture line for the skin closure. In utilizing cartilage, one should carve the implant so that it will fit fairly accurately into the depression to be filled in. The demand for adequate cover by subepithelial tissues is the same as that when using fascia as an implant. The postoperative dressing should be very firm. It may be of plaster, lightly applied, or it may consist of a wetted starch bandage.

The consideration of depressed scars related to orbital fractures is the third subdivision of types previously considered as probable. In these instances the technique is to use cadaver cartilage or autogenous cartilage as plaques or wedges. In the consideration of these cases it is assumed that the fracture has been healed for so long that the open reduction of the fracture is an impossibility. Figure 11 illustrates a simple type. The patient had a depressed fracture of the floor of the orbit with a depressed scar at the rim of the orbit and with diplopia due to the depressed eyeball. In

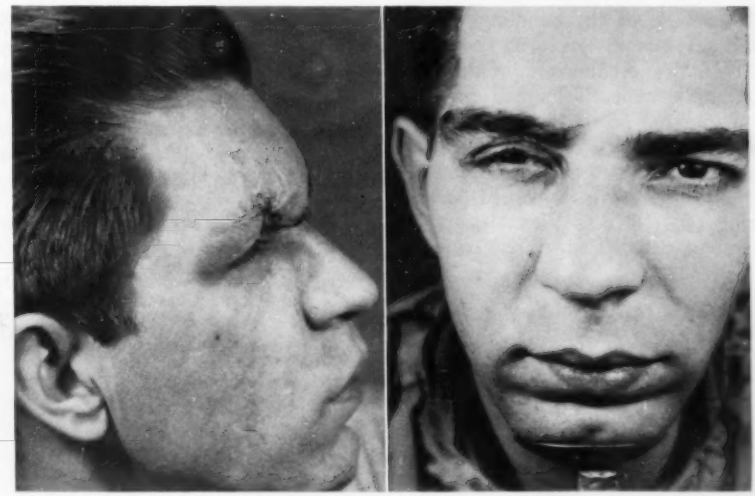

FIG. 10—(Left) Oblique, adherent, depressed scar of the rim of the orbit, which crosses the internal canthal angle and the bridge of the nose. (Right) After cadaver cartilage implantation and after the scar resection

this case a wedge-shaped scale of cartilage, cadaver or autogenous, was to be placed beneath the eyeball along the floor of the orbit to elevate the eyeball to a proper level. If autogenous cartilage is used, as it was in this case, the graft should be inserted beneath the periosteum of the remaining floor. Cadaver cartilage should be placed upon the surface of the periosteum. A second flat plate of cartilage is then placed anterior and at a right-angle to this, forming a capital T upon its side, to fill in the depression resulting from the posterior displacement of the bone fragments.

Another of our cases offers an even

FIG. 11—(Left) Depressed scar from fracture of orbit. (Right) Cartilage correction. The line across both parts of the figure emphasizes the relative position of the eyeball before and after the operation.

better example of the handling of depressed scars from fractures about the orbit (fig. 12). At the time of the first visit, the patient had a marked cicatrix, with depression of the orbital rim and of the malar bone (fig. 12, A). The scar was resected and two large pieces of cadaver cartilage were implanted before suturing (fig. 12, B). When the operative wound had healed, it was evident that too much cartilage had been placed in position. Another incision was made, the anterior surface of the cartilage, still in position, was shaved off to reduce the thickness of the implant, and the operative incision was closed. The result was satisfactory (fig. 12, C).

In another instance it was necessary not only to reset the scar and to graft

FIG. 12—Original defect, a marked cicatrix with flattened malar and maxilla from displaced malunited fractures. (A) The position of the eyeball before and after the operation. (B) After scar resection, resuture and cadaver cartilage grafts. (C) End result.

FIG. 13—(A) Adherent, depressed scar from fracture of orbit, with loss of the orbital rim and a portion of the floor of the orbit. (B) Immediate result following pedicle flap and cartilage implant. Incision on chest; suture line at site of cartilage graft—autogenous cartilage was used. (C) Intermediate result. (D) The condition upon discharge, after the secondary suture lines had been trimmed.

cartilage, but also to replace soft tissues (fig. 13). In this instance the pedicle flap was formed from above and the cartilage graft was done simutaneously (fig. 13, B). It is possible that cadaver cartilage might have been as satisfactory, but autogenous cartilage was used in this case for many good reasons. A free skin graft, however, was contraindicated because of the depth and amount of cicatrix present; it did not form a satisfactory bed for a free skin graft.

CONCLUSION

In the consideration of the correction of depressed scars, the following points are re-emphasized: a careful and meticulous study of the defect present; careful preoperative planning for scar resections, for incision lines, and for the replacement of tissues, if this is necessary; and the meticulous closure of the operative wounds in layers, according to their normal positions and relationships.

LACERATIONS INVOLVING THE BULBAR CONJUNCTIVA AND THE EYEBALL

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INVOLVEMENT of the globe frequently accompanies lacerations of the eyelid and the orbit. However, the globe may be injured without any laceration of the neighboring tissues. Lacerations of the conjunctiva are rather simple to handle unless they involve deeper structures or are associated with infection. Ordinarily conjunctival lacerations are only sutured when they are ragged, U-shaped, or the conjunctiva is reflected. When the conjunctiva is badly lacerated, a running suture is the best type to use. Do not use interrupted sutures because their knots cause irritation and discomfort.

Lacerations of the global wall are often accompanied by lacerations of the cornea or the sclera, lacerations of both with prolapse of the uvea or with loss of the intraocular fluid, especially the vitreous. Traumatic cataract or retinal detachment may complicate the treatment of the original laceration. Other complications arise from intraocular foreign bodies, such as wood, grit, stone, steel, brass and, in recent years, a good deal of glass. Some of these foreign substances need special attention if they are chemically active. If they are small and chemically inactive, such as shattered glass, removal may be unnecessary.

A perforation of the globe requires immediate attention in order to prevent further prolapse of viterous and other internal tissues of the eye. An immediate evaluation of the situation is necessary, followed by prompt surgical repair. When caring for global injuries it is important to know the extent of the patient's vision, although sometimes it is impossible to ascertain.

The physician must determine the extent of trauma to the various tissues, both to the global wall and to the contents. This can be done with the loupe, with the ophthalmoscope, and with the slit lamp, which should be set up near the emergency room.

Roentgenograms are important, but can be postponed until an emergency operation is done. The presence of a foreign body can be determined, to some extent, by the history. An emergency radiologist may or may not be available at night. Ocular contents may be lost while the roentgenogram is being made.

Some 150 years ago, ocular lacerations were repaired by direct suture. Seventy-five years ago physicians began to repair corneal lacerations with the conjunctival flap. Today, with improved suture material, improved forceps, and improved diagnostic material, physicians are able to evaluate the extent of the laceration, and have gone back to the direct suture method of repair.

The conjunctival flap was used, in some instances, to repair a corneal laceration complicated by prolapse of the iris. The iris was abscised, and a conjunctival flap was put over it and held with mattress sutures at both sides. I do not recommend this method today for several reasons. When treated early, the laceration can be sutured with direct suture, and the surgeon can be more certain of a watertight anterior chamber.

Condensation of a commentary on slides.

Presented at the Sixty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 14-19, 1956, Chicago.

Sutures can be preplaced and left untied before iris abscision. The surgeon then frees the iris from the lateral walls and closes the anterior chamber, having previously performed a toilet of the wound. In some instances it is necessary to stroke the iris pillars into position by making a lateral keratotomy 5 mm. to the side or below, inserting a narrow spatula into the opening, and sweeping the iris pillars back into position. In some instances if these patients are cared for early, before the iris adheres to the cornea, it is possible to sweep the entire herniated iris back into position, restore the pupil shape, and then close the anterior chamber, filling it with either saline solution or air.

An extensive, zigzagging laceration of the cornea is impossible to repair with a conjunctival flap because not only does the conjunctival flap permit gaping beneath it, but a conjunctival flap will never secure the iris and hold it back. It will not seal the anterior chamber; furthermore, granulation tissue and deep scar tissue extend into the anterior chamber. Finally, the eyeball is destroyed as a functioning organ.

One patient suffered a laceration of the globe and within twenty-four hours had panophthalmitis. Anything that I could do was useless and he lost his eye because of the severe infection.

Another laceration that had previously been repaired with a conjunctival flap resulted in a deformed pupil which adhered to the ectatic corneal scar. In such instances, it is necessary to free the iris from the scar. That can be done either by an excision of the piece of ectatic cornea, or by freeing the iris and approximating the margins of the excised region by direct suture.

In the case of a ragged laceration, when an accurate approximation is essential, the surgeon will probably use the new atraumatic sutures. I prefer to use stainless Grieshaber needles with 6-0 black silk. If the cornea is bisected, I place three sutures; then after replacing the iris, I place two intermediate sutures: one between the first and second, and another one between the second and third. The iris is kept back by keeping the anterior chamber filled with a stream of normal saline solution. Finally an air bubble is instilled into the anterior chamber.

Sometimes a surgeon prefers to overlay a conjunctival flap, but in my experience it is neither necessary nor advisable.

Corneal sutures should be of very fine silk. They may be left in for a period of twelve to sixteen days. In one case, after excision of an ectatic corneal scar with incarcerated iris, the astigmatism which had been present was reduced, but unfortunately some photophobia persisted.

One patient was treated for a scleral laceration and an intraocular foreign body with herniated vitreous. The splinter was removed and the vitreous abscised. The sclera was sutured first, and then the conjunctiva was repaired.

Subconjunctival hemorrhage is an early clue to ocular scleral laceration.

To repair a scleral laceration put sutures in halfway through its thickness, excise the prolapsed tissue, and then close the scleral sutures tight. When suturing sclera it is important to overlay with conjunctiva. The conjunctiva is a rather elastic membrane, and one can draw it across and attach it about 4 mm. to one side of the sclera. This provides a double wall of protection against infection entering the globe.

Frequently, we are faced with a problem of adequate examination in marked cases of edema. Examination in such cases is difficult. Sometimes, it is almost impossible to separate the lids to inspect

the globe well. I have found that injection of hyaluronidase, 20 units per cubic centimeter, into the edematous area, will frequently reduce edema but not hematoma.

Another technique for reducing the board-like edema is the intravenous administration of ACTH by the drip method. Twenty-five to 40 units of ACTH in one-half liter of dextrose, given by slow drip, will, sometimes within twelve hours, reduce the edema considerably.

Sequelae of these globe lacerations include massive hemorrhage, for which I have no treatment, and occasionally retinal detachment. A perforation in the sclera posterior to the ora serrata is repaired by interrupted sutures followed by diathermy of the bared sclera to seal any retinal tear that may exist. One of the frequent complications in these cases is the formation of vitreous bands and the danger of subsequent retinal detachment. It is possible that implantation of vitreous may help. I have used scissors to divide these bands, but that method is not very effective in letting the retina go into place.

The management of the traumatic cataract is usually given delayed consideration. Mydriasis is essential.

The prognosis is guarded in all ocular lacerations, except the ones that simply puncture the cornea and do not traumatize the deeper tissues. Infection must be handled by the adequate use of sulfonamides, oral antibiotics, and penicillin, given subconjunctivally (5,000 units in 1 cc. of water). The patient or his relatives should be questioned about the patient's sensitivity to penicillin; if not sensitive, large doses of penicillin can be continued for several days, or he can be given chloromycetin in massive doses. The latter gives a high concentration in the intraocular fluids more readily than some of the other antibiotics.

Local or general anesthesia may be employed for the repair of global lacerations as indicated. Topical anesthesia is sufficient for the conjunctival lacerations. The intravenous injection of 250 to 500 mg. of Diamox thirty to sixty minutes before the operation reduces intraocular pressure. For penetrating lacerations one must also use hyaluronidase and Novocain retrobulbarly to soften the globe. If the perforation is only scleral, it is often wise to do a paracentesis of the cornea in order to further soften the globe and prevent additional loss of vitreous while the wound is being sutured.

Both eyes should be kept bandaged for two weeks if retinal detachment is threatened, then both may be unbandaged and stenopeic spectacles used.

DISCUSSION

BYRON C. SMITH, M.D.
WENDELL L. HUGHES, M.D.
NEW YORK, NEW YORK

DR. SMITH: I agree with 98 per cent of everything that has been said. I wish to compliment the speakers on their wonderful presentations and their beautiful demonstrations. Dr. Spaeth mentioned in his paper the use of cadaver cartilage, a process which has been used in New York during the last five years. We have found that it absorbs and is quite unsatisfactory. We prefer autogenous bone or cartilage. It may be that the cadavers in Philadelphia are more healthy than those in New York.

Dr. Spaeth also spoke about the complete excision of scar tissue. At times I think it is advantageous to leave scar tissue in a wound for the purpose of building up a depression. If one excises excessive tissue, it causes further depression of the surface scar. I have found that the superficial tissues can be satisfactorily advanced over the scar tissue.

In Dr. Ruedemann's presentation, I was interested in the case with the beer cap in the maxillary sinus, and I wonder if that bottle cap went through the floor of the orbit. I am interested in this aspect because it pertains to a paper that I shall present later concerning internal orbital fracture. I am sure that had roentgenograms been taken before the laceration was sutured, the cap probably would have been shown. If one has the opportunity and available roentgenray equipment, presurgical pictures should be taken. In the late treatment of infected wounds, I think it is advisable to take cultures and perform drug sensitivity tests at the time of the operation, so that administration of the specific drug may be started as soon as possible.

Dr. Hartman did not emphasize the necessity for eradication of lacrimal infection. In one of the cases illustrated, a swelling toward the medial canthus might have been a lacrimal infection. I dare say it wasn't, because Dr. Hartman is not the type of surgeon who would operate in its presence. However, lacrimal infection should be either treated at the time of the operation or eliminated before definitive surgery is started.

Dr. Hartman also showed an instance in which a full-thickness graft from the retroauricular angle was taken to replace skin of the upper lid. Since the patient refused the use of donor skin from the opposite upper lid, Dr. Hartman's alternative was a wise choice. Thin skin is superior to supraclavicular-triangle skin. A split-skin graft over a conformer may be used in such cases. This may be done without lid adhesions.

I have very little to add to Dr. Thorpe's presentation. I am very pleased to know that he no longer uses the conjunctival flap and is now directly suturing the cornea and replacing the iris when it is at all feasible.

DR. HUGHES: This has been a wonderful evening and we have a lot of information that we can take home with us. Dr. Spaeth emphasized some important points, one in particular about hemostasis among the deeper tissues. Make sure that the patient doesn't have deep bleeding that will continue and stretch the sutured wound apart. The deep tissue closure that he emphasized, I think, is particularly important.

I agree with what Dr. Smith has said about cartilage. Personally, I don't like to use preserved cartilage too well. If there is any question of viability, the autogenous cartilage is preferred and is not difficult to obtain. It can be taken from the synchondrosis of the sixth and seventh ribs down near the lower border of the ribs where they join the sternum.

I agree with Dr. Smith that roentgenograms should be taken before you operate to show up bottle caps and other foreign bodies. Dr. Ruedemann emphasized early repair in these cases, and I think that point is very well taken. If you can't repair them the first day, the patient is very frequently out of shock the second day. There is a great need for education; the general surgeon and the plastic surgeon must be shown the advantages of having eye injuries taken care of as soon as possible, and they should be encouraged to call in the ophthalmologist at an early stage.

Dr. Ruedemann showed slides of a group of injuries he has encountered, although I certainly don't see many like them. His lacrimal canaliculus reconstruction is interesting. I think it is quite possible, in the early stage, to rejoin the lower canaliculus when it is cut across, but I haven't had much success in getting a final functioning result. I want to be able to put a drop of colored medicine,

such as mercurochrome, in the eye and have it appear spontaneously in the nose; I don't call it a satisfactory result if you must put a cannula into the canaliculus in order to squeeze some fluid down into the nose; I want that eye free from tearing, with spontaneous drainage into the nose.

One thing that was not mentioned in any of the presentations is aftercare—the immediate aftercare including the dressings that are put on at the time of operation. I think that in many of the cases that were illustrated here there was edema preoperatively; it is very difficult to work with edematous tissue. You can prevent edema, and control postoperative edema, by the application of a moderate, not extreme, pressure dressing. That pressure dressing will also remove some of the swelling present at the time of the operation and will help to place and keep the tissues in proper position until primary healing has taken place.

Lost time frequently results in infection and swelling, which add to the difficulty of repair.

Lacrimal infection was discussed by Dr. Smith. I think that there are two methods of handling that problem. If you feel that there is no chance of re-establishing drainage, the lacrimal sac can be removed. I hesitate to do that unless it is absolutely necessary. I would rather open the sac into the nose in order to drain any infection away from the operative area. That is quite feasible and can be done well.

I agree entirely with Dr. Smith's choice of tissues for repair of lid skin areas, upper or lower, that are near the lid margin. The upper lid skin from the opposite lid is by far the best tissue to use. As a second choice, split skin is much better than any full-thickness graft because the texture, thickness, wrinkling, general contour, and color are far better. Supra-auricular skin may be used if necessary. It must be used as a split-skin graft.

Dr. Hartman brought out the problem of the contracture of late scars. That brings up the general physiology of healing when there is swelling with exudation and transudation into the tissues nearby which interferes with primary surgical repair. If you do not use some sort of pressure dressing after the repair, transudation is almost inevitable. Swelling will tear away some of the original suture lines, widen suture lines, and increase the deep granulation tissue, the subsequent fibrous tissue, and the cicatricial tissue that is formed during the healing process. Healing also brings up the question of keloid. If a proper amount of pressure is used to prevent swelling and infiltration of the tissues, it will prevent keloid development.

It is not always possible to correct complicated cases in one procedure. It is frequently necessary to do more than one procedure, sometimes five or six, to get a fully satisfactory result. The remark was made in one of the papers—I think by Dr. Hartman—that the canaliculus was hopelessly lost in scar tissue. Now it is very often possible to dissect part of the scar tissue and find the end of the canaliculus that is joined to the sac. You can often get an estimate of how much canaliculus is left in contact with the sac by seeing how much canaliculus there is next to the punctum. See how far your probe will go into the punctum. If it will go in only one or two millimeters, then you may assume that there is a fairly good piece of the lacrimal canaliculus in contact with the sac. Often you can find that piece and bring it out into the conjunctival sac where it will be in contact with the tears and provide fairly good drainage by re-establishing a punctum.

Occasionally a conjunctivodacryocystostomy may be necessary. Dr. Hartman brought up the question of the displacement of the medial canthus by laceration and the replacement of it by surgery; he mentioned wire suture. I know that a good many men do use wire sutures, but personally I cannot see that a permanent type of suture is at all necessary. I think that a suture that will last for four or five weeks, such as a heavily chromicized gut suture, is all that is necessary. After that length of time a wire suture remains, with no retaining effect, and simply acts as a foreign body. I occasionally use wire to bind some bony fragments together when I feel that other sutures will not suffice.

Lid adhesions comprise another very important subject brought out by Dr. Hartman. I think that they serve well to reduce late cicatricial displacement resulting from contracture of the lids postoperatively.

Dr. Thorpe brought up some interesting points, and I agree with most of what he says. The question of whether to use silk or gut in some corneal lacerations is important. I like the new 6-0 plain gut on the extremely sharp micropoint needles. I use it in a mattress suture through the lips of the wound, bring each arm over to the conjunctiva just beyond the limbus, and anchor the knot in the conjunctiva rather than putting it over the cornea. The extra trauma to the cornea, caused by the knot, is obviated. I think that

that type of suturing works very well in almost any laceration whenever two corneal edges are to be joined together. Dr. Thorpe mentioned the technique of putting a bubble of air in the anterior chamber. He did not mention that it is important to have the patient put his head in such a position that the air bubble is kept at the part of the anterior chamber angle where it is needed to separate the iris from the cornea. If the patient has a laceration of the temporal side, fairly low, goes back to bed and lies with that side of the head down, the air bubble will go to the nasal side of the anterior chamber and will be ineffective in separating the cornea and the underlying structures. If the air bubble rests behind the laceration while the aqueous is re-forming, it will almost always prevent synechias.

The very small, new type of half circle needles is extremely valuable in sewing up lacerations that are fairly far back in the sclera. Anterior lacerations in the sclera don't offer too much of a problem. Dr. Thorpe mentioned the subconjunctival injection of penicillin. In addition to that, I use a subconjunctival injection of aerosporin with neomycin, which is quite effective in preventing pyocyaneus infection. Fifty thousand units of penicillin in .25 cc. of saline solution can be obtained very readily. Add .25 cc. of 2 per cent procaine hydrochloride because that concentration of penicillin is quite painful when it is injected. The penicillin-procaine is injected underneath the conjunctiva close to the sclera to produce a good concentration of the antibiotic inside the eye both in the aqueous and in the vitreous. Do this immediately after the operation when there are very few organisms present. At the same time, but in another area, inject 50,000 units of aerosporin in .05 cc. of saline solution and .05 cc. of procaine hydrochloride. A tuberculin syringe is best for this small injection. The 50,000 units of the aerosporin and 50,000 units of penicillin form a very effective guard against intraocular infection. I have been using penicillin since 1942 and added the aerosporin three years ago. I have used this combination in every case in which any instrument has entered the globe or whenever the globe has been opened, even if it was only for paracentesis, and there has not been one postoperative intraocular infection in several thousand cases.

Dr. Thorpe also mentioned atropine. I think that it is very important to limit the movement of the ciliary body and the choroid, but if the wound is near the periphery of the cornea, I imagine that Dr. Thorpe would use something to pull the iris away from the periphery in addition to the injection of the air. The location of a laceration in the eye is quite important. If the laceration is over the ciliary body, and the ciliary body is protruding, sympathetic ophthalmia becomes a potentiality. We must always keep that potential danger in mind, even though it does not happen very often. When it does occur, it is frequently disastrous. Of course the steroids are fairly effective agents which prevent the development of sympathetic ophthalmia and even combat it when it has already developed.

I think that this has been a perfectly wonderful evening. I wish to thank all of the speakers for their fine presentations. They have spent a tremendous amount of time gathering this material, and we have all benefited greatly from it.

TRANSACTIONS

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15 Second Street S.W., Rochester, Minnesota

INDUSTRIAL OPHTHALMOLOGY AND OTOLARYNGOLOGY

A workable method of estimating loss of efficiency in industry is necessary for proper settlement of cases involving compensation awarded by industrial commissions and claims for impairment of function as a result of conditions of employment. The amount of the award is determined by a formula which is applied by a lay group, such as a state agency or commission, or a jury which may or may not seek expert medical opinion on interpretation of the evidence. Since the formula must cover all degrees and types of loss, there must be a standard normal efficiency level presumably applicable to everyone working under present-day industrial conditions and recognizing industrial hazards that are variable as preventive and safety measures are developed. Physical and sensory efficiency of individuals is relative to standards set up to apply to various groups such as age, sex, geographical areas, climate, occupation, and many other factors which in themselves change with the times. For instance, an increase in average longevity and the employment of more people of advanced years requires re-examination of sensory and physical norms, particularly of seeing and hearing. Continuous research in these fields is now supported by the Academy along with funds from a number of sources, and the results are frequently the subject of discussion in our scientific assemblies.

In this issue of the TRANSACTIONS appears such an article: *Estimation of Loss of Visual Efficiency* by Dr. Edmund B. Spaeth, consultant in ophthalmology to the Council on Industrial Health of the American Medical Association. The Joint Committee on Industrial Ophthalmology of the American Medical Association and the Academy was dissolved in 1955 in the interests of better administration, and since then the four national ophthalmological societies have been requested to nominate consultants as advisors to the Council on Industrial Health. Dr. Spaeth has served on the Joint Committee and as a consultant to the Council and has been in close touch with the medical and social problems in industry. The American Medical Association's Council on Indus-

trial Medicine will consider suggestions from all other societies and will administer whatever programs are developed in the industrial fields of vision and hearing.

The estimation of loss of earning ability through reduction in vision and hearing, whether the result of natural processes of ageing or of conditions of employment, is a problem that requires reexamination as requirements in various skills are altered in conformance with modern industrial processes and the installation of automation in replacing manual labor. Presbyopia and presbycusis, along with scotoma, insular deafness, and other functional defects, must be arbitrarily standardized so that terms and their definition are understood by all who must consider the fair administration of justice in compensation and liability cases. The public will expect the medical profession to set up standards and formulas for estimation of loss of ability on a graduating scale. The problem is medical, legal, and social and must be solved by men of broad thinking as well as technical ability.

LETTERS TO THE EDITOR

September 17, 1957

Dear Dr. Benedict:

We are greatly interested in locating experimental subjects with proven total loss of function of both inner ears. We have written to a number of otologists without success and are wondering if it might be possible to place a notice in your journal which might read as follows:

The U. S. Naval School of Aviation Medicine is greatly interested in locating persons who have total loss of function of both inner ears either through bilateral labyrinthectomies or bilateral VIIIth nerve sections. These patients will be used in studies on the human centrifuge and rotational devices at Pensacola. There is no hazard and little or no discomfort. Healthy subjects between the ages of 18 and 50 can be used and will be paid $15 per day plus expenses.

The cooperation of any physicians in helping locate such persons will be greatly appreciated. Please address correspondence to: Captain Ashton Graybiel, MC, USN; Director of Research; Naval School of Aviation Medicine; Naval Air Station; Pensacola, Florida.

We will be glad to pay any charges in connection with this notice but it will be necessary that we receive a bill prior to date of publication.

Thanking you in advance for any attention you give to this matter, I remain

Yours sincerely,

[Signed] ASHTON GRAYBIEL
Captain, MC, USN

September 23, 1957

Dear Doctor Benedict:

Dr. Donald Shafer first reported the use of vitreous in the treatment of retinal detachment in New York in 1955; we have been interested in this subject since that time. We have used vitreous in a number of cases of retinal detachment, but since our vitreous is obtained from postmortem material, the possibility of contamination has given us some concern.

During the past year we have been carrying out a series of experiments in which we have inoculated vitreous with various organisms and storing it at 4° C. as recommended by Dr. Shafer. We have found that dog and rabbit vitreous have virtually no antibacterial properties against the organisms used under

these conditions, but human vitreous is usually self-sterilizing. In fact, several specimens of vitreous which were badly infected when first obtained have become sterile with storage.

It seems that the rate of self-sterilization varies with the type and concentration of the organism inoculated. We have experimented with six common organisms and the only one of the six which human vitreous appears to be unable to destroy under these conditions is the spore former, B. subtilis. Our findings, therefore, fully support those of Dr. Shafer; the observations of Dr. Suie and Dr. Sroufe do not necessarily conflict with Dr. Shafer's and ours; because we have found that several days may elapse before some organisms disappear from the vitreous. We agree with Dr. Suie and Dr. Sroufe, however, that vitreous is best collected and stored using sterile technique throughout.

Our experiments have been delayed by the difficulty in obtaining fresh human vitreous, but we are hoping to publish a full report in the not too distant future.

Yours sincerely,

[Signed] HOWARD REED
[Signed] JOHN C. WILT
[Signed] GEORGE TUSHINGHAM

Department of Bacteriology and Ophthalmology

University of Manitoba and Winnipeg General Hospital

Winnipeg, Manitoba

In Memoriam

William Wightman Blair.....Pittsburgh, Pennsylvania
October 25, 1957

Carl F. Breisacher.....Charleston, West Virginia
October 16, 1957

Moses Lewis Breitstein.....Baltimore, Maryland
July 20, 1957

Linus S. Cave.....Syracuse, New York
August 10, 1957

Clarence Charles Hetzel.....Ogden, Utah
August 8, 1957

Walter Jellis.....West Somerville, Massachusetts
May 12, 1957

Simon Jesberg.....Los Angeles, California
June 24, 1957

Hobart Clemens Johnson.....Mankato, Minnesota
July 23, 1957

Frank Conrad Keil.....New York, New York
October 15, 1957

Heinrich Günther Kobrak.....Clarkston, Michigan
October 9, 1957

Albert N. Lemoine, Sr.....Kansas City, Missouri
August 30, 1957

James Prince Lewis.....Boston, Massachusetts
May 31, 1957

Harold Irving Lillie.....Rochester, Minnesota
August 27, 1957

Julian Barnett Marks.....Los Angeles, California
April 5, 1957

Roy David Martin.....Grand Island, Nebraska
September 18, 1957

Frederic J. McIntire.....Lynn, Massachusetts
June 18, 1957

H. Edward Miller.....St. Louis, Missouri
August 3, 1957

Ralph Alex Poirier.....Birmingham, Michigan
September 28, 1957

George F. Reineke.....New Ulm, Minnesota
May 21, 1957

Edward Daniel Risser.....Pasadena, California
May 1, 1957

Albert Eugene Seligman.....Staten Island, New York
June 17, 1957

Ferris N. Smith.....Grand Rapids, Michigan
September 18, 1957

Clarence Archibald Veasey, Sr.....Spokane, Washington
August 11, 1957

Peter De Tilius Westerhoff.....Midland Park, New Jersey
June 21, 1957

Charles Augustus Young.....Roanoke, Virginia
July 25, 1957

INTERNATIONAL ASSOCIATION OF SECRETARIES OF OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETIES

Oto-rhino-laryngo-ophthalmology

It seems superfluous to stress the need for cooperation between ophthalmology and otolaryngology, but it is necessary to point out that this cooperation is decreasing more and more in some countries.

Let us look backward for a moment to the time of large and fruitful development of closely related studies and activities of ophthalmologists and otolaryngologists. We cannot help but express our dissatisfaction about the present state of affairs for only very few papers appear concerning oto-rhino-ophthalmology. Very little mutual work is done, and scant pertinent knowledge is available in either of these fields pertaining to joint problems. Therefore, there exists many areas, scientific as well as clinical, which await satisfactory solutions: for example, the question of physiological, pathological and clinical neurovegetative connection between eye, nose and ear.

Almost every day we are in need of consultation with an otolaryngologist. He does as well as he can. But both the ophthalmologist and the otolaryngologist need to know their reciprocal clinical needs and the respective problems. Without this knowledge, cooperation will often remain unfruitful.

Apart from the present need we must consider another aspect of the situation. Our younger colleagues did not practice during the profitable period of joint oto-rhino-laryngo-ophthalmologic studies. They must be made conscious of the value of reciprocal clinical cooperation.

It is easy to see why this separation of scientific work has taken place. Counter measures ought to be taken. I propose that a preliminary discussion on this subject be made during a meeting of the European cooperative directors and the officers of the International Association of Secretaries of Ophthalmological and Otolaryngological Societies. The officers will be in Europe for the Eighteenth International Congress of Ophthalmology, to be held in Brussels, October 8-12, 1958. Afterwards, suggestions submitted during this discussion should be elaborated upon and their execution arranged at the annual meeting of our Association in the United States in 1959.

From a still larger point of view it is to be hoped that the possibilities will be explored of publishing a textbook of diseases which will interest, at at the same time and in many ways, eye, and ear, nose and throat specialists alike. The most eminent scientists of every country should pour into this work their entire knowledge and their lifetime of clinical experience, freely, fully, and without prejudice. Only the wording of chapters should be left to individual authors. The over-all execution should be under the supervision of the American Academy of Ophthalmology and Otolaryngology and of the Council of our Association. Such a textbook will give the young, and also the older, ophthalmologists and otolaryngologists a precious stimulus to attempt the development of more and more intelligent cooperation, and this is the main purpose of our Association. The task, although arduous, is worthy of every effort.

E. Filippi Gabardi
Honorary Secretary General
European Society of Ophthalmology

Necrology

IN MEMORIAM

October 1956 - October 1957

John Hedley Atkinson, Watertown, N. Y.
Robert Beattie, Clare, Mich.
John Wallace Beil, Kansas City, Mo.
William Wightman Blair, Pittsburgh, Pa.
Truman Leo Boyes, New York City, N. Y.
Joseph Bruder, New York City, N. Y.
Abram Breneman Bruner, Cleveland, Ohio
Frank E. Burch, St. Paul, Minn.
George Winston Burchfield, Maryville, Tenn.
Robert Caldwell, Little Rock, Ark.
Gail Ellsworth Chandler, Miami, Fla.
Joseph Daniel Collins, Northampton, Mass.
James Sumner Davis, New York City, N. Y.
Lyn Waller Deichler, Philadelphia, Pa.
Murray McColl Dewar, Grand Rapids, Mich.
William Edwin Dickerson, Danville, Va.
James Arthur Downing, Des Moines, Iowa
Octavus Dulaney, Louisville, Ky.
Slocomb R. Edwards, El Centro, Calif.
Clarence William Engler, Cleveland, Ohio
John Walker Fairing, Greensburg, Pa.
Guy Rothwell Fisher, Staunton, Va.
William Howard Folsom, Fond du Lac, Wis.
Leonard Fox, Detroit, Mich.
Robert Carson Fraser, Los Angeles, Calif.
Milton Tacitus Gaillard, Baldwin, N. Y.
Samuel Goldhammer, Denver, Colo.
Thomas John Goodfellow, Saratoga Springs, N. Y.
James Dudley Goodman, Elyria, Ohio
George F. Gracey, Harrisburg, Pa.
Herbert Graebner, New York City, N. Y.
Richard Gillespie Hamilton, Pittsburgh, Pa.
Halvor Larson Harley, Pleasantville, N. J.
Solomon Mayer Hartzell, Beverly Hills, Calif.
Arthur Merl Hauer, Columbus, Ohio
James Alexander Henderson, St. Louis, Mo.
Carl Henning, Rehoboth Beach, Dela.
Clarence Charles Hetzel, Sr., Ogden, Utah
Harvey James Howard, Clearwater, Fla.
Ray Whitbeck Hughes, Lake Worth, Fla.
Thomas Edmond Hughes, Delaplane, Va.
Harry James Inglis, Newton, Mass.
William R. Jacobs, Lewiston, Idaho
Mark Walton Jacoby, Cleveland, Ohio
Walter Jellis, West Somerville, Mass.
Simon Jesberg, Los Angeles, Calif.
Harry Vincent Judge, Albany, N. Y.
Henry Kapp, Haverhill, Mass.
Percival Kaufman, New York City, N. Y.
Samuel Newton Key, Austin, Texas
LeRoy Taylor Kincannon, Birmingham, Ala.
Charles Royal King, Toledo, Ohio
George Madison Krieger, Michigan City, Ind.
Nathan Kent Lazar, Miami Beach, Fla.
Lavel H. Leeson, Vancouver, B. C., Canada
Forrest Shetterly LeTellier, Knoxville, Tenn.
James Prince Lewis, Boston, Mass.
Joseph Davidson Lewis, Santa Barbara, Calif.
Harold Irving Lillie, Rochester, Minn.
Hugh Montgomery Lokey, Atlanta, Ga.
Samuel Herman Long, Chattanooga, Tenn.
Julian Barnett Marks, Los Angeles, Calif.
Roy David Martin, Grand Island, Neb.
James Ernest McAskill, Watertown, N. Y.
Frederic J. McIntire, Jr., Lynn, Mass.
O. B. Monosmith, Lorain, Ohio
Lewis Francis Morrison, San Francisco, Calif.
John Sanford Moyer, Metuchen, N. J.
Eugene Lee Myers, St. Louis, Mo.
Harley Dore Newby, Rapid City, S. D.
John Henry Ohly, Brooklyn, N. Y.
Richard Johnson Payne, St. Louis, Mo.
Louis M. Pearlman, New York City, N. Y.

Francis Peter Quinn, Dubuque, Iowa
George F. Reineke, New Ulm, Minn.
George Bond Ridout, LaCrosse, Wis.
Otto Carl Risch, Flushing, N. Y.
Edward D. Risser, Pasadena, Calif.
Kersey Clyde Riter, Salt Lake City, Utah
Daniel Abell Russell, San Antonio, Texas
Albert Eugene Seligman, Staten Island, N. Y.
Edward Cecil Sewall, Palo Alto, Calif.
Francis Xavier Siegel, Cincinnati, Ohio
Morris Bigelow Simpson, Kearney, Mo.
Ferris N. Smith, Grand Rapids, Mich.
Frank R. Spencer, Boulder, Colo.
Abraham Bennett Spiegelglass, River Edge, N. J.
William F. C. Steinbugler, Brooklyn, N. Y.
William Herman Stokes, Lake City, Mich.
Gilbert Stevens Tabor, Millerton, N. Y.
George Holten Traugh, Fairmont, Pa.
Harry Allen Van Osdol, Carmel, Ind.
Clarence Archibald Veasey, Spokane, Wash.
James Washington Ward, Greenville, Texas
Joseph Roosevelt Weintrob, Atlantic City, N. J.
Peter De Tilius Westerhoff, Midland Park, N. J.
Minor Elisha White, Kankakee, Ill.
Charles B. Wylie, Columbus, Ohio
Herman Eugene Yazel, Kansas City, Mo.
Arthur Meyer Yudkin, New Haven, Conn.

JOHN HEDLEY ATKINSON

Dr. John Hedley Atkinson was born in Parrsboro, Nova Scotia, on June 29, 1888. He received his preliminary education in Cascadilla, Nova Scotia, Ithaca, New York, and Wooster, Ohio. His degree in medicine was received from McGill University, and postgraduate work was done at Harvard University.

Dr. Atkinson began the practice of medicine in Watertown, New York, in 1915. He was chief of the Department of Otolaryngology at Mercy Hospital in Watertown, and attending otolaryngologist at the House of Good Samaritan Hospital in Watertown.

Among his professional affiliations were memberships in the American Academy of Ophthalmology and Otolaryngology, the American College of Surgeons, and state and local medical societies.

Dr. Atkinson died in Watertown on November 26, 1951. He is survived by his wife, Florence Atkinson; a daughter, Mrs. John Logan; and a son, John H. Atkinson, Jr.

ROBERT BEATTIE

Dr. Robert Beattie was born in Fergus, Ontario, Canada, on November 2, 1870. He received his preliminary education in Palmerston, Ontario, and the degree of Doctor of Medicine was conferred upon him by the Detroit College of Medicine (now Wayne University College of Medicine) in 1903. Postgraduate work was done abroad in London, England, and Vienna, Austria.

Dr. Beattie began the practice of medicine in Detroit, Michigan, in 1903. He was on the consulting staff of Providence Hospital, Mt. Carmel Hospital, and Eloise Hospital in Detroit and Wayne. From 1920 until 1930 he served as associate professor of ophthalmology at the Detroit College of Medicine.

Dr. Beattie had been a Fellow of the American Academy of Ophthalmology and Otolaryngology since 1906, and had been made a Life Fellow in 1936. He was also a Fellow of the American College of Surgeons and a member of the American Medical Association and of state and county medical societies. He was an honorary member of the Detroit Ophthalmological Society, the Detroit Otolaryngological Society, and the Detroit Ophthalmological Club.

Dr. Beattie died on August 18, 1956, in Clare, Michigan. He is survived by a son, Robert Beattie, Jr.; a daughter, Lillis Beattie Saffell; and a grandson, Robert Beattie, III.

JOHN WALLACE BEIL

Dr. John Wallace Beil, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died in Kansas City, Missouri, on September 22, 1956.

Dr. Beil was born in Manhattan, Kan-

sas, on November 10, 1875. He attended the Kansas State Agricultural College for one year, and received his degree as Doctor of Medicine from the University Medical College, Kansas City, Missouri, in 1901. He did his postgraduate work as assistant to Dr. T. B. Tiffany. From the time of his graduation until his retirement, he engaged in the practice of his specialty in Kansas City, Missouri.

Dr. Beil was a diplomate of the American Board of Ophthalmology. In addition to his fellowship in the American Academy of Ophthalmology and Otolaryngology, he held memberships in the Kansas City Eye, Ear, Nose & Throat Society and in state and local medical societies.

A son, Dr. Wallace C. Beil of Las Vegas, New Mexico, who is also a Fellow of the Academy, is his sole survivor.

WILLIAM WIGHTMAN BLAIR

Dr. William W. Blair was born in Elizabeth, Pennsylvania in 1866. In 1889 he was graduated from Hahnemann Medical College and Hospital of Philadelphia with the Doctor of Medicine degree. He interned at Pittsburgh Homeopathic Hospital and later was certified a specialist by the American Board of Ophthalmology. He was on the staff of Pittsburgh Eye and Ear Hospital from 1909 to 1937, serving as chief of staff from 1930 to 1937. He was professor of ophthalmology at the Clinic of Pittsburgh from 1909 to 1935, and was on the staff of St. Margaret Memorial Hospital. At the time of his death on June 12, 1957, Dr. Blair was professor emeritus of ophthalmology at the University of Pittsburgh School of Medicine.

Dr. Blair founded the Lippincott Library, and started an isolation ward at Pittsburgh Eye and Ear Hospital. He also was one of the founders of the Pennsylvania Association for the Blind.

Dr. Blair is survived by one daughter and three sons.

TRUMAN LEO BOYES

Dr. Truman Leo Boyes was born in Glanford, Ontario, Canada, on June 15, 1898. He was the son of Dr. Edward T. Boyes, who was a pupil of Osler, and Mrs. Amelia Tanner Boyes. Much of his early childhood was spent in the country and in the company of his father who made house calls by means of horse and buggy. This experience gave him an early interest in medicine, which remained with him to the last.

After attending public schools of Glanford, he completed his preparatory education in 1915 at St. Mary's Catholic High School in Hamilton, Ontario. He entered the University of Toronto in 1916 and received his medical degree in 1922. Following an internship in the Hamilton General Hospital, he was accepted for ophthalmic training at Bellevue and Long Island College hospitals. He entered practice in association with the late Dr. Clyde E. McDannald in New York in 1927.

Dr. Boyes was appointed to the staff of the New York Eye and Ear Infirmary at the time he entered practice, and soon became the chief of Dr. McDannald's service. He developed an early interest in muscular anomalies and surgery. He was director of the Orthoptic Clinic and liaison surgeon for resident training.

For many years, Dr. Boyes taught courses in the Postgraduate School of the New York Eye and Ear Infirmary and became a clinical professor of ophthalmology in the postgraduate division of the New York University Bellevue Medical College. Not only was he an inspiring leader in his clinic, but he was also a convincing lecturer and teacher in the American Academy of Ophthalmology and Otolaryngology for many years.

His hospital connections included New York Eye and Ear Infirmary, and consulting appointments to Misericordia, St. Mary's of Hoboken, New Jersey, and Good Samaritan hospitals of New York.

Dr. Boyes was a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology. He served as chairman of the Section of Ophthalmology of the New York Academy of Medicine during 1953-54, and was an honorary member of the New York Eye and Ear Alumni Association. He was a fellow of the American College of Surgeons and of the New York Academy of Medicine. He also held membership in the Pan-American Association of Ophthalmology, the New York Ophthalmological Society, the Physicians' Scientific Society, the Celtic Medical Society, the American Medical Association, and state and local medical societies. He was a diplomate of the American Board of Ophthalmology.

Dr. Boyes died on October 17, 1956, in New York City. He is survived by his mother, Mrs. Amelia A. Boyes; a brother, J. Webster Boyes, and three sisters, Mrs. Charles Higgins and Misses Kathleen and Monica Boyes.

JOSEPH BRUDER

Dr. Joseph Bruder, who was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died in New York City on August 18, 1956.

Dr. Bruder was born in Hungary in December 1874, and received his preliminary education at the Otolaryngological Academy in Vienna, Austria. His degree as Doctor of Medicine was received from Bellevue Hospital College of Medicine in New York in 1898, and he returned to Vienna for his postgraduate work. During his active practice, he was associated with the New York City Hospital and Mount Sinai Hospital, but he had been retired for many years.

Dr. Bruder is survived by his wife and two daughters.

ABRAM BRENEMAN BRUNER

Dr. Abram Breneman Bruner, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died in Cleveland, Ohio, on January 9, 1957.

Dr. Bruner, who was born in Columbia, Pennsylvania, on October 13, 1891, received his premedical education at the Wesleyan University of Connecticut. His degree as Doctor of Medicine was obtained from Columbia University College of Physicians and Surgeons in 1916, and postgraduate work in ophthalmology was done at the Manhattan Eye and Ear Hospital.

Dr. Bruner began the practice of medicine in Cleveland, Ohio, in 1921. Beginning as a demonstrator of ophthalmology in the medical department of Western Reserve University, he rose to the position of associate clinical professor of ophthalmology in that institution. At the time of his death he held the position of professor emeritus in ophthalmology. He served as associate ophthalmologist at the Lakeside Hospital and Dispensary in Cleveland.

In addition to his affiliation with the American Academy of Ophthalmology and Otolaryngology, Dr. Bruner was a member of the American Ophthalmological Society and of the Association for Research in Ophthalmology. He was also a Fellow of the American College of Surgeons. He had served as president and vice-president of the Academy of Medicine of Cleveland, and was a trustee and past president of the Cleveland Medical Library. Dr. Bruner was a diplomate of the American Board of Ophthalmology.

Although Dr. Bruner was fond of fishing and had a large collection of etchings, his chief hobby was collecting stamps pertaining to medicine. He was a member of the American Society of Philatelists, and won many citations and medals for his collection.

Dr. Bruner is survived by his wife, two sons, and two daughters.

FRANK E. BURCH

(See Eulogy in the TRANSACTIONS, July-August 1956, following p. 534.)

GEORGE WINSTON BURCHFIELD

Dr. George W. Burchfield was a junior member of the American Academy of Ophthalmology and Otolaryngology. He was born September 1, 1894 at Maryville, Tennessee, and was graduated from Vanderbilt University School of Medicine at Nashville in 1917 with the degree of Doctor of Medicine. He was certified a specialist by the American Board of Otolaryngology, and was in private practice, owning a five-bed hospital for treatment of eye, ear, nose, and throat diseases in Maryville, until his death at Blount Memorial Hospital on February 26, 1957.

Dr. Burchfield was a member of the Tennessee Academy of Ophthalmology and Otolaryngology, Blount County Medical Society, Tennessee State Medical Society, and American Medical Association.

ROBERT CALDWELL

Dr. Robert Caldwell, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died in Little Rock, Arkansas, on December 3, 1956.

Dr. Caldwell was born in Martinsville, Indiana, on October 28, 1876. He attended Central Normal College in Danville, Indiana, and received his degree as Doctor of Medicine from the Hospital College of Medicine in Louisville, Kentucky,

in 1901. Postgraduate work in his specialties was done in Chicago, Philadelphia, New York, London, Berlin, and Vienna.

Dr. Caldwell began the practice of medicine in 1901 in Flat Rock and Brownstown, Indiana. In 1921 he joined the faculty of the University of Arkansas School of Medicine, affiliating with the Department of Otolaryngology. He served as chief of staff of St. Vincent Infirmary from 1927 to 1931. He was also on the staff of Baptist Hospital. For many years he was a member of the board of the Arkansas Hospital for Nervous Diseases, and was president of the board from 1932 to 1936. He was the first doctor in Arkansas to do work in bronchoscopy.

In addition to his fellowship in the American Academy of Ophthalmology and Otolaryngology, Dr. Caldwell was a Fellow of the American College of Surgeons, and a member of the American Medical Association and of state and local medical societies. He was a past president of the Arkansas Medical Society, and served as president of the Pulaski County Medical Society a number of times.

In 1947, the College of the Ozarks conferred upon Dr. Caldwell the honorary degree of Doctor of Science.

Dr. Caldwell is survived by his wife, Monta Almerath Caldwell, and a brother and two sisters.

GAIL ELLSWORTH CHANDLER

Dr. Gail Ellsworth Chandler was born in Carman, Illinois, on August 2, 1893. He received his degree as Doctor of Medicine from Jefferson Medical College in Philadelphia, in 1918, and had one year of postgraduate work in France before serving in World War I as a Lieutenant Commander.

In 1925, Dr. Chandler began the practice of otolaryngology in Miami, Florida, where he served on the staffs of Jackson Memorial Hospital and Victoria Hospital.

A diplomate of the American Board of Otolaryngology, Dr. Chandler was a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology, and a Fellow of the American College of Surgeons. He was a member of the American Medical Association and of state and local medical societies.

On December 15, 1956, Dr. Chandler died in Miami, Florida. None of his family survives.

JOSEPH DANIEL COLLINS

Dr. Joseph Daniel Collins, aged 75, died January 13, 1957, at Northampton, Massachusetts. He was a graduate of Harvard Medical School, Boston, and a member of the American Academy of Ophthalmology and Otolaryngology, and the American Medical Association. He was on the staffs of the Veteran's Administration Hospital, Northampton State Hospital, and the Cooley Dickinson Hospital. Dr. Collins also served in World War I.

JAMES SUMNER DAVIS

Dr. James S. Davis was born in Boston, Massachusetts, on June 22, 1913. He graduated with honors from Boston University, and received his degree as Doctor of Medicine from Boston University Medical School in 1939. His postgraduate work in otolaryngology was done at Beth Israel Hospital in Boston and at Jersey City Medical Center.

Dr. Davis began the practice of otolaryngology in South Norwalk, Connecticut, in October 1937. He served as senior attending physician at the Norwalk General Hospital and was director of the Eye, Ear, Nose & Throat Department of the same hospital.

Dr. Davis was a diplomate of the Board of Otolaryngology. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons. He also held membership in the American Medical Association and in state and local medical societies.

Dr. Davis died in New York City on September 30, 1955. He is survived by his wife, Florence Josem Davis, and one daughter, Joan Susan.

LYN WALLER DEICHLER

Dr. Lyn Waller Deichler, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died in Philadelphia on October 2, 1956.

Dr. Deichler was born in Lancaster, Pennsylvania, on January 18, 1882. He received his degree as Doctor of Medi-

cine from the University of Pennsylvania Medical School in 1903. He had been consulting ophthalmologist at Douglas Hospital, Belmont Hospital, Salvation Army Home and Hospital, and the Merciful Savior Home for Crippled Children. He was an active consultant in ophthalmology at Philadelphia General Hospital.

In addition to his fellowship in the American Academy of Ophthalmology and Otolaryngology, Dr. Deichler was a Fellow of the College of Physicians of Philadelphia, and a member of the American Medical Association and of state and local medical societies. He was a diplomate of the American Board of Ophthalmology, having taken his examinations in 1934.

Dr. Deichler is survived by his son, John W. Deichler, M.D., who is also a Fellow of the Academy.

MURRAY McCOLL DEWAR

Dr. Murray McColl Dewar was born in Cedar Springs, Michigan, on January 28, 1897. He received his degree as Doctor of Medicine from Wayne University School of Medicine in Detroit in 1920, and his postgraduate work in ophthalmology was done in Vienna, London and New York.

Dr. Dewar began the practice of medicine in Grand Rapids, Michigan, in 1926. He served on the staff of Blodgett, Butterworth, and St. Mary's hospitals.

A diplomate of the American Board of Ophthalmology, Dr. Dewar was a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology. He also held membership in the American Medical Association and in state and local medical societies.

Dr. Dewar died in Grand Rapids on March 30, 1957. He is survived by his wife and two sons, Murray McColl, Jr., and John David.

WILLIAM EDWIN DICKERSON

Dr. William Edwin Dickerson, who was born in Appomattox, Virginia, on September 19, 1898, died in Danville, Virginia, on March 28, 1957.

Dr. Dickerson was a graduate of the University of Richmond, and received his degree as Doctor of Medicine from the Medical College of Virginia in Richmond, in 1922. He began the practice of medicine in Princeton, West Virginia in 1924. In 1929, he entered New York Polyclinic Hospital for a residency in otolaryngology, and since 1930 he had limited his practice in Danville, Virginia, to that specialty. He was a member of the staffs of Memorial Hospital and of Danville Community Hospital.

Dr. Dickerson was a diplomate of the American Board of Otolaryngology, a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the Virginia Society of Ophthalmology and Otolaryngology, the Southern Medical Society, the American Medical Association, and state and local medical societies.

Dr. Dickerson is survived by his wife and one son, a student at the Medical College of Virginia.

JAMES ARTHUR DOWNING

Dr. James Arthur Downing was born in Exeter, Nebraska, on February 23, 1887. He received his degree as Doctor of Medicine at the Medical College of Drake University in 1909, and took postgraduate work in otolaryngology under the late Dr. Harris P. Mosher of Boston, and special work in broncho-esophagoscopy under Chevalier Jackson of Philadelphia.

During his entire professional life Dr. Downing engaged in the practice of medicine in Des Moines, Iowa. When his son, Dr. Arthur H. Downing, started the practice of ophthalmology, Dr. James limited his practice to otolaryngology. He was chief of the Eye, Ear, Nose & Throat Department at Iowa Methodist Hospital; on the consulting staff of Broadlawns Polk County Hospital; and on the courtesy staff of Iowa Lutheran Hospital and Mercy Hospital. During World War I he served as first lieutenant in the medical corps. He felt that his main contribution to his specialty had been the development of a technique using a three-way tube for closed circuit oxygen supply for tracheotomy patients. This was devised during the polio epidemic of 1953.

A diplomate of the American Board of Otolaryngology, Dr. Downing was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Chest Physicians. He also held membership in the Pan-American Broncho-Esophageal Society, the International Broncho-Esoph-

ageal Society, the American Broncho-Esophagological Association, the International Congress of Otolaryngology, the Iowa Academy of Ophthalmology and Otolaryngology, the Des Moines Academy of Ophthalmology and Otolaryngology, the American Medical Association, and state and local medical societies. He was treasurer of the Iowa State Medical Society from 1942 to 1946. In 1943 he served as President of the Polk County Medical Society and as a Trustee of the Society for several years.

Dr. Downing died in Des Moines on December 14, 1956. He is survived by his wife, Gertrude Herrmann Downing, and three sons, James Hillis Downing, of Lyford, Texas; Dr. William L. Downing, of Jamestown, North Dakota; and Dr. Arthur H. Downing, of Des Moines, who is also a Fellow of the Academy.

OCTAVUS DULANEY

Dr. Octavus Dulaney, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died in Louisville, Kentucky, on June 17, 1956.

Dr. Dulaney was born in Harrisville, Mississippi, on July 11, 1878. He received his degree as Doctor of Medicine at the University of Nashville in 1901, and became associated with his father in the practice of medicine in Crystal Springs, Mississippi, the same year. About 1915 he established and built a clinic in Dyersburg, Tennessee, which later became the Baird-Brewer General Hospital. He later moved to Louisville, where he was affiliated with General Hospital, Kentucky Baptist Hospital, Norton Memorial Infirmary, and St. Anthony Hospital. For several years he served on the faculty of the University of Louisville Medical School in the Department of Otolaryngology.

In addition to his fellowship in the American Academy of Ophthalmology and Otolaryngology, Dr. Dulaney was a Fellow of the American College of Surgeons, and a member of the Kentucky Eye and Ear Society, the Louisville Eye and Ear Society, the Southern Medical Association, the American Medical Association, and state and local medical societies. He was a past president of the Tennessee Medical Association.

Dr. Dulaney is survived by his wife and one son.

SLOCOMB R. EDWARDS

Dr. Slocomb R. Edwards, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died in El Centro, California on December 27, 1954, at the age of 68. He had lived and practiced ophthalmology and otolaryngology in South Bend, Indiana, until 1935, when he moved to El Centro.

Dr. Edwards had received the degree of Doctor of Medicine from the University of Maryland School of Medicine in 1908.

CLARENCE WILLIAM ENGLER

Dr. Clarence William Engler died in Cleveland, Ohio, on February 23, 1957.

Dr. Engler was born in Fremont, Ohio, on August 1, 1895. He was a graduate of Adelbert College and received his degree as Doctor of Medicine from Western Reserve Medical School in 1919. As a protege of Dr. Secord H. Large, Dr. Engler decided to enter the practice of otolaryngology, and did postgraduate work in Cleveland, Philadelphia, Boston, Vienna, and Edinburgh. After 1923, his practice in Cleveland was restricted to his specialty.

Dr. Engler was appointed to the staff of St. Vincent Charity Hospital in 1935, and he also served on the staff of Cleveland City Hospital. He was assistant clinical professor of otolaryngology at the Medical School of Western Reserve University.

Dr. Engler was a diplomate of the American Board of Otolaryngology. At the time of his death, he was president of the American Broncho-Esophagological Society. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons, and held membership in the Cleveland Otolaryngological Club, the Cleveland Academy of Medicine, the Cleveland Medical Library, the American Medical Association, and state and local medical societies. He had been a trustee of the Cleveland Medical Library, and for nine years had been president of the staff of St. Vincent Charity Hospital.

Surviving Dr. Engler is one sister, Mrs. Philip Gottron, of Fremont, Ohio.

JOHN WALKER FAIRING

Dr. John W. Fairing died at the age of 84 at College Hill Hospital, Dayton, Ohio.

Dr. Fairing was graduated from Baltimore Medical College in 1898. He became an associate member of the American Medical Association, was certified a specialist by the American Board of Ophthalmology and the American Board of Otolaryngology, was an inactive member of the American Academy of Ophthalmology and Otolaryngology, was a member of the Medical Society of the state of Pennsylvania, and was a fellow of the American College of Surgeons. Dr. Fairing practiced in Greensburg, Pennsylvania, where he was on the staff of the Westmoreland Hospital, and lived at Shaker Heights, Ohio.

GUY ROTHWELL FISHER

Dr. Guy Rothwell Fisher was born in New Hope, Virginia, on October 16, 1889. He attended the Medical College of Virginia, where he received the degree of Doctor of Medicine in 1912. Following his graduation, he began the practice of general medicine in his home community. In 1920, he entered postgraduate work in otolaryngology at Columbia University Medical School.

Dr. Fisher became a diplomate of the American Board of Otolaryngology, and had practiced his specialty in Staunton, Virginia, since completing his residency. He was a member and past president of the staff of King's Daughters' Hospital in Staunton.

In addition to fellowship in the American Academy of Ophthalmology and Otolaryngology, Dr. Fisher had served as president and secretary of the Virginia Society of Ophthalmology and Otolaryngology, and as president of the Medical Society of Virginia, the Augusta County Medical Association, and the Medical Society of the Valley of Virginia. He was a member of the American Medical Association, the Southern Medical Association, and the Tri-State Medical Society.

Dr. Fisher died in Staunton, Virginia, on January 15, 1957.

WILLIAM HOWARD FOLSOM

Dr. William H. Folsom, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, was born on October 7, 1870, in Dodge County, Wisconsin. His death occurred on February 1, 1957, at Fond du Lac.

Dr. Folsom's career began with education at Wayland Academy and Hilldale College. He then received his degree as Doctor of Medicine from Rush Medical School in 1897, at which time he began private practice in Markeson, Wisconsin. He was on the staff of St. Agnes Hospital, Fond du Lac.

In addition to his membership in the Academy, Dr. Folsom was a member of the Wisconsin-Upper Michigan Society of Ophthalmology and Otolaryngology and an honorary member of the Wisconsin Medical Society and the American Medical Association.

During his many years of devoted medical service to his fellow man, Dr. Folsom found time to help found the Green Lake Bible Institute. He loved the study and care of native trees and planted many along the shores of Green Lake. He was extremely interested in other civic affairs and organizations. The love and patience he showed to his patients and co-workers was something that will not be forgotten.

Dr. Folsom is survived by one daughter, Mrs. G. A. Cooper of Madison, Wisconsin, and two sons, Dr. Hugh Folsom of Framingham, Massachusetts, and Howard Folsom of Manitowish Waters, Wisconsin.

LEONARD FOX

Dr. Leonard Fox was born in Toronto, Ontario, Canada, on March 22, 1923. He attended the University of Michigan and received his degree as Doctor of Medicine from the same university in 1945. His residency in ophthalmology was taken at the City of Detroit Receiving Hospital, and he began the practice of his specialty at Wyandotte, Michigan, in 1951.

Among Dr. Fox's professional affiliations were staff appointments at Wyandotte General Hospital, Detroit Memorial Hospital, Oakwood Hospital in Dearborn, Michigan, and Sinai Hospital in Detroit. He was a member of the Department of Ophthalmology at Wayne University.

Dr. Fox was a diplomate of the American Board of Ophthalmology. He was a

Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the Detroit Ophthalmological Society, the Detroit Surgical Society, the American Medical Association, and state and local medical societies.

Dr. Fox died in Detroit, Michigan, on October 7, 1956. He is survived by his wife.

ROBERT CARSON FRASER

Dr. Robert Carson Fraser died December 13, 1956, at the age of 72 years.

In 1905 Dr. Fraser had graduated from Detroit College of Medicine. He was certified by the American Board of Otolaryngology, and then served on the staffs of St. Joseph Hospital in Burbank and the Presbyterian Hospital-Olmsted Memorial in Los Angeles.

Dr. Fraser was a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology, a fellow of the American College of Surgeons, and a member of the American Medical Association and of state and local medical societies.

MILTON TACITUS GAILLARD

Dr. Milton T. Gaillard of Baldwin, New York, died October 25, 1956, at the age of 52 years. In 1932 he was graduated by McGill University Faculty of Medicine, Montreal, Canada. He was certified a specialist by the American Board of Ophthalmology. He served on the staffs of South Nassau Communities Hospital in Oceanside, Mercy Hospital in Rockville Centre, Nassau Hospital in Mineola, and Meadowbrook Hospital in Hempstead.

Dr. Gaillard was a Junior Member of the American Academy of Ophthalmology and Otolaryngology, and a Fellow of the American College of Surgeons.

SAMUEL GOLDHAMMER

Dr. Samuel Goldhammer was 68 years of age when he died May 22, 1957, of cerebral thrombosis. He had lived in Denver, Colorado all of his life. In 1910 he was graduated from Denver and Gross College of Medicine, and was later on the faculty of the University of Colorado School of Medicine. Dr. Goldhammer was on the staffs of Beth Israel Hospital, National Jewish Hospital, and the General Rose Memorial Hospital. He was a Junior Member of the American Academy of Ophthalmology and Otolaryngology.

THOMAS JOHN GOODFELLOW

Dr. Thomas John Goodfellow was born in Parham, Ontario, Canada on February 11, 1884. He was graduated from the Queens University in 1907 and received his degrees as Doctor of Medicine and as Master of Surgery from his alma mater in 1909. His postgraduate work in ophthalmology and otolaryngology was done at the State Hospital for Insane at Warren, Pennsylvania, and at the Post Graduate Hospital in New York City.

The entire professional life of Dr. Goodfellow, from 1915 until his death on January 19, 1957, was spent in Saratoga Springs, New York, where he served as chief of the Eye, Ear, Nose and Throat Department of Saratoga Hospital and as consultant of the Benedict Memorial Hospital, in Ballston Spa, New York.

Dr. Goodfellow was a diplomate of the American Board of Ophthalmology and of the American Board of Otolaryngology. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons, and held membership in the Association for Research in Ophthalmology, the Pan-American Association of Ophthalmology, the Pan-American Association of Otolaryngology, the American Trudeau Society, the American Public Health Association, the American Society for the Advancement of Science, the World Medical Association, the Biological Photographic Association, the National Society for the Prevention of Blindness, the New York Academy of Science, the American Medical Association and state and local medical societies. He was a past president of both the Saratoga Springs and the Saratoga County Medical Societies.

At the time of his death, Dr. Goodfellow had almost completed a history of the Saratoga County Medical Society, which was to be presented at an exhibit at the State Medical Society Meeting in New York City in the spring of 1957.

Dr. Goodfellow is survived by his wife, Nell Virginia Goodfellow; and twin daughters, Mrs. Andrew McNaughton, and Mrs. Duncan Fraser.

JAMES DUDLEY GOODMAN

Dr. James Dudley Goodman, who died on February 14, 1957, in Elyria, Ohio, was born in Cleveland, Ohio, on December 19, 1912. After studying at Western Reserve University in Cleveland and Ohio State University in Columbus, Ohio, Dr. Goodman received his degree as Doctor of Medicine from Ohio State University School of Medicine in 1939.

In 1942, Dr. Goodman began the general practice of medicine in Cleveland, Ohio. Since 1951, when he moved to Elyria, Ohio, he had confined his practice to the specialty of ophthalmology.

Dr. Goodman was a diplomate of the American Board of Ophthalmology. In addition to his fellowship in the American Academy of Ophthalmology and Otolaryngology, he was a member of the Cleveland Ophthalmology Club, of the American Medical Association, and of state and local medical societies.

As a hobby, Dr. Goodman was interested in radio. He was an amateur radio operator of Station W-8EHQ of Elyria.

Dr. Goodman is survived by his wife, Hope Goodman, two sisters, and three brothers.

GEORGE F. GRACEY

Dr. George F. Gracey, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died in Harrisburg, Pennsylvania, on January 2, 1957.

Dr. Gracey was born on September 9, 1874, in Altoona, Pennsylvania. He was graduated from Millersville Normal High School in 1901, and matriculated at the University of Pennsylvania where he received his degree as Doctor of Medicine in 1908.

After studying in Germany and Austria, Dr. Gracey became professor of chemistry at the University of Texas Medical School in Galveston. In 1913 he began a residency in otolaryngology at the New York Eye and Ear Infirmary and was graduated in 1916. When he began the practice of his specialty in Plainfield, New Jersey, this practice was interrupted by World War I in which Dr. Gracey served with the Army Medical Corps in the United States and in France. At the close of the war he resumed his practice in Harrisburg where he was an active member of the staff of the Harrisburg Hospital until 1939, and on the consulting staff until his death.

In addition to his fellowship in the American Academy of Ophthalmology and Otolaryngology, Dr. Gracey was a Fellow of the Pennsylvania Academy of Ophthalmology and Otolaryngology, and a member of the American Medical Association and of state and local medical societies.

Dr. Gracey is survived by his wife, Jean Smith Gracey.

HERBERT GRAEBNER

Dr. Herbert Graebner was born in 1890 at Milwaukee, Wisconsin.

He was certified a specialist in otolaryngology by the American Board of Otolaryngology in 1933. He served as Assistant Surgeon at Manhattan Eye, Ear, Nose, and Throat Hospital and the New York Hospital in New York City; was Assistant Visiting Otolaryngologist at Willard Parker and St. Vincent's Hospitals, and was an instructor in otolaryngology at Cornell University Medical College.

Dr. Graebner was a Fellow of the American Academy of Ophthalmology and Otolaryngology, a member of the American Laryngologic, Rhinologic, and Otolaryngologic Society, the New York County Medical Society, the New York State Medical Society, and the American Medical Association.

Dr. Graebner had been totally disabled since 1949, and died July 17, 1956, in New York City.

RICHARD GILLESPIE HAMILTON

Dr. Richard Gillespie Hamilton was born in Roswell, New Mexico, on December 12, 1908. He was a graduate of the University of Pittsburgh and received the degree of Doctor of Medicine from the University of Pittsburgh Medical School in 1933.

During his entire professional life, Dr. Hamilton engaged in the practice of medicine in Pittsburgh, where he was an active member of the staffs of the Eye and Ear Hospital, St. Francis General Hospital and Magee Hospital. He was an associate professor of otolaryngology at his alma mater.

A diplomate of the American Board of Otolaryngology, Dr. Hamilton was a Fellow of the American Academy of Ophthalmology and Otolaryngology, the American College of Surgeons, and the Pennsylvania Academy of Ophthalmology and Otolaryngology. He was a member of the Board of Directors of the latter society. Dr. Hamilton was also a member of the American Society of Ophthalmologic and Otolaryngologic Allergy, the Pittsburgh Allergy Society, the Pittsburgh Academy of Medicine, and of the American Medical Association and state and local medical societies.

Dr. Hamilton died at his home, Fairway Lane, Fox Chapel, on May 22, 1957. He is survived by his wife and two sons.

HALVOR LARSON HARLEY

Dr. Halvor Larson Harley was born in Laurelton, New Jersey, on July 18, 1882. He received his degree as Doctor of Medicine from the Medico-Chirurgical College of Philadelphia, now the University of Pennsylvania Medical School, in 1908. Postgraduate work in ophthalmology was done at the University of Pennsylvania Medical School in 1919.

Dr. Harley was chief of the Eye Service at Atlantic City Hospital from 1924 until his retirement in 1952. He also served as consultant for Children's Seashore House and for the Betty Bacharach Home for Afflicted Children in Longport, New Jersey.

Dr. Harley was a specialist certified by the American Board of Ophthalmology. He was a fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the Association for Research in Ophthalmology, of the American Medical Association and of state and local medical societies. He had served as historian of the Atlantic County Medical Society.

Dr. Harley was the author of two books of poetry, "Words and Music," and "Late Harvest." Among his other interests were reforestration and an apiary. He had a rare collection of etchings and wood cuts.

Dr. Harley died in Pleasantville, New Jersey, on May 16, 1957. He is survived by his wife, Alice R. Harley; a daughter, Elisabeth, who is an orthoptic technician in Durham, North Carolina; and a son, Robison D. Harley, who is also a Fellow of the Academy.

SOLOMON MAYER HARTZELL

Dr. Solomon M. Hartzell was born in 1879. He received the degree of Doctor of Medicine in 1901 from the University of Pennsylvania School of Medicine.

Dr. Hartzell was a Fellow of the American Academy of Ophthalmology and Otolaryngology, of the International College of Surgeons, and of the American College of Surgeons.

On September 10, 1956, Dr. Hartzell died at his residence in Beverly Hills, California.

ARTHUR MERL HAUER

Dr. Arthur Merl Hauer, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died in Columbus, Ohio, on September 15, 1956.

Dr. Hauer, who was born in Bucyrus, Ohio, on August 28, 1881, received his degree as Doctor of Medicine from Ohio Medical University in Columbus in 1906, and engaged in the practice of otolaryngology in Columbus from 1910 until his death. He was formerly on the faculty of the Ohio State University College of Medicine and then served on the staffs of the Grant and White Cross hospitals in Columbus. Due to illness, he had been semi-retired since 1951.

In addition to his fellowship in the American Academy of Ophthalmology and Otolaryngology, Dr. Hauer was a fellow of the American College of Surgeons, and a member of the Columbus Academy of Medicine, of the American Medical Association, and of state and local medical societies.

None of his family survive.

JAMES ALEXANDER HENDERSON

Dr. James Alexander Henderson was born in Brooklyn, New York, on September 19, 1907. He attended Washington & Lee University in Virginia, and received his degree as Doctor of Medicine from George Washington University in Washington, D. C., in 1934. He served as a resident in the Brooklyn Eye and Ear Hospital from 1936 to 1938. Following his residency, he served as ship's surgeon aboard commercial liners. This work was interrupted by participation in World War II. Following his tour of duty, he began the practice of oto-

laryngology in Quincy, Illinois, where he served on the staffs of St. Mary's Hospital and Blessing Hospital.

Dr. Henderson was a diplomate of the American Board of Otolaryngology. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons, and a member of the Central Illinois Society of Ophthalmology and Otolaryngology, of the American Medical Association and of state and local medical societies.

Dr. Henderson died in St. Louis, Missouri, on September 2, 1956. He is survived by his wife, Matilda Babinsky Henderson; and two daughters, Ann Louise and Stacey Alexandra.

CARL HENNING

Dr. Carl Henning, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died at his home in Rehoboth Beach, Delaware, on September 12, 1956.

Dr. Henning, who was born in Beaver Falls, Pennsylvania, in 1880, grew up in Fargo, North Dakota. He received his undergraduate education at the University of Michigan, and his degree as Doctor of Medicine was conferred upon him by the George Washington University School of Medicine, Washington, D. C., in 1905. Following a year of postgraduate study in Vienna, Austria, he returned to Washington to begin the practice of ophthalmology. He served on the staffs of the Episcopal Eye, Ear and Throat Hospital and of Providence Hospital for many years. In 1953, he retired to Rehoboth Beach, Delaware, where he engaged in limited practice.

In the early days of Dr. Henning's practice, he studied under and was associated with Dr. William Holland Wilmer, the founder of Wilmer Institute of Johns Hopkins University.

Dr. Henning was a past president of the Clinical Club of Washington, D. C. He was a member of the Medical Society of the District of Columbia, Section of Ophthalmology, and of the American Medical Association.

Dr. Henning is survived by his wife, Marion Dilcer Henning, and a sister, Mrs. George Fillius.

CLARENCE CHARLES HETZEL, SR.

Dr. Clarence Charles Hetzel, Sr., was born in Avoca, Iowa, on May 10, 1877 and died August 7, 1957, in Ogden, Utah.

Dr. Hetzel received his Bachelor of Arts degree in 1899 and the degree of Doctor of Medicine in 1903 from the University of Iowa. He served his internship at the University of Iowa Hospital and was associated with Dr. L. A. Dean in 1903 and 1904. He completed postgraduate work and an internship at Wills Eye Institute, Philadelphia, in 1904 and 1905. From 1906 to 1911 Dr. Hetzel engaged in the practice of ophthalmology and otolaryngology in Davenport, Iowa. He then moved to Ogden, Utah where he established his practice and served as consultant for the Union Pacific Coal Mines. Dr. Hetzel was an original member of the staff of the Thomas Dee Memorial Hospital in Ogden.

Dr. Hetzel was a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology and a member of the Pan-American Association of Ophthalmology. He had served on the State Examining Board, and he was a captain in the Medical Corps during World War I.

In July 1917 Dr. Hetzel married the former Alta Smith, who survives with sons, Dr. C. Charles Hetzel, Jr., Ogden, who is also a Fellow of the Academy, and Robert S. Hetzel, Costa Mesa, California, and one daughter, Mrs. Mary F. Fisher, Ogden.

HARVEY JAMES HOWARD

Dr. Harvey James Howard was born in Churchville, New York, on January 30, 1880. He was graduated from the University of Michigan in 1904, and received his degree as Doctor of Medicine from the University of Pennsylvania in 1908.

Following his graduation from the University of Pennsylvania, Dr. Howard went to Canton, China, where he was head of the Department of Ophthalmology at the University Medical College until 1913, ophthalmologist at the Canton Christian College and ophthalmic surgeon at the Canton Hospital until 1915. From 1916 until 1918, he was a Fellow of the China Medical Board of the Rockefeller Foundation at Harvard, where he received the Master of Arts degree in 1918. He returned to China as professor

and head of the Department of Ophthalmology at Union Medical College in Peking where he served until 1927. During this time he spent a year in postgraduate study in Vienna, Austria, and served as medical advisor to the Department of Aeronautics of the Chinese government.

In 1927 Dr. Howard became professor and head of the Department of Ophthalmology at Washington University School of Medicine in St. Louis, where he was chief of the ophthalmic service at Barnes Hospital, and at St. Louis Children's and St. Louis Maternity hospitals, ophthalmologist at the Evangelical Deaconess Hospital, and a member of the staff of the Morton F. Plant Hospital. He had been an executive director of the Oscar Johnson Institute of Research in Ophthalmology and Otolaryngology in St. Louis, and was a former director of the Department for the Prevention of Blindness of the Missouri Commission for the Blind. In 1950 Dr. Howard retired to Clearwater, Florida, where he engaged in a restricted practice until a year before his death on November 6, 1956.

Dr. Howard was a diplomate of the American Board of Ophthalmology. He was a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology, a Fellow of the American College of Surgeons, and he also held membership in the American Ophthalmological Society and in the Association for Research in Ophthalmology. He was an associate member of the American Medical Association.

During World War I while Dr. Howard was serving as captain in the Medical Corps of the United States Army, he devised a depth perception test which has continued in official use for selection of aviation personnel in the armed services. Dr. Howard was the author of a book, "Ten Weeks With Chinese Bandits," which recounts the history of his captivity by bandits in Heilung-Chiang Province, China, during the summer of 1925.

Surviving Dr. Howard are his wife, Alice Tilson Howard; two daughters, Mrs. Margaret Jackson of Bucks County, Pennsylvania, and Mrs. Martha Blake of New York City; and one son, Brigadier General James H. Howard of Washington, D. C.

RAY WHITBECK HUGHES

Dr. Ray Whitbeck Hughes was born in Augusta, Michigan, on July 29, 1890. He received his degree as Doctor of Medicine from the Detroit College of Medicine and Surgery (now Wayne University College of Medicine) in 1916, and following his internship he served in the Medical Corps of the 32nd Division until the close of World War I.

In 1919 Dr. Hughes became affiliated in the practice of otolaryngology with Dr. Harold Wilson of Detroit, and served on the staffs of Harper, Grace, and Florence Crittenton hospitals. In 1945 he was forced to retire on account of his health. He moved to Lake Worth, Florida, where he was engaged in a limited practice. In Lake Worth, he was a member of the staffs of St. Mary's and Good Samaritan hospitals.

Dr. Hughes was a diplomate of the American Board of Otolaryngology. He was a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Medical Association, of Wayne County Medical Association, and of Palm Beach County Medical Association.

Dr. Hughes died in Lake Worth on August 28, 1956. He is survived by his wife, Bertha C. Hughes, and one son.

THOMAS EDMOND HUGHES

Dr. Thomas Edmond Hughes, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died at his farm, "Aspindale," Delaplane, Fauquier County, Virginia, on April 2, 1957.

Dr. Hughes was born at Amissville, Rappahanock County, Virginia, on January 20, 1884. He attended Randolph-Macon Academy and was a graduate of Richmond College. He received his degree as Doctor of Medicine from the University of Virginia, Charlottesville, in 1910. Postgraduate work in otolaryngology was done at New York Eye and Ear Infirmary and at Manhattan Eye and Ear Hospital in New York. He began the practice of his specialty in 1918 in Richmond, Virginia, where he was on the staffs of St. Luke's Hospital, Johnston-Willis Hospital, the hospital of the Medical College of Virginia, Sheltering Arms Hospital, and Retreat for the Sick.

He had retired from practice in Richmond in 1953, and moved to his farm at

Delaplane. He opened an office at Front Royal, Virginia, on a restricted schedule, but at the time of his death his practice had become so heavy that he could no longer handle it alone.

Dr. Hughes was a specialist certified by the American Board of Otolaryngology in 1926. He was a member of the American Laryngological, Rhinological and Otological Society, of the Richmond Eye, Ear, Nose and Throat Society, of the Richmond Academy of Medicine, of the American Medical Association, and of state and local medical societies.

As hobbies, Dr. Hughes farmed and raised horses and fine cattle.

Dr. Hughes is survived by four brothers and one sister.

HARRY JAMES INGLIS

Dr. Harry James Inglis was born in Middletown, Connecticut on July 2, 1880.

Dr. Inglis received his degree of Doctor of Medicine from Harvard University in 1904, and became consulting surgeon at the Boston Dispensary at Malden Hospital, Malden, Massachusetts. At the time of his death on August 23, 1956, he was professor emeritus in otolaryngology at Tufts College Medical School, Boston.

Dr. Inglis was certified as a specialist in otolaryngology by the American Board of Otolaryngology in 1925, and was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology. He was a fellow of the American College of Surgeons, and a member of the American Medical Association, the American Laryngological, Rhinological, and Otolaryngological Society, and the New England Oto-Laryngological Society.

WILLIAM RIGBY JACOBS

Dr. William R. Jacobs was born September 21, 1906 in Ogden, Utah. He graduated from Rush Medical College, Chicago in 1937.

Dr. Jacob's private practice was interrupted by military service in the medical corps of the U. S. Air Force from 1943 to 1946. He was a flight surgeon and parachutist, and was separated from the Force with the rank of Captain. He received the Air Medal with three oak leaf clusters.

Certified as a specialist by the American Board of Ophthalmology, Dr. Jacobs practiced ophthalmology in Lewiston, Idaho, where he was chairman of the ophthalmological section of St. Joseph's Hospital.

Dr. Jacobs was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Medical Association and of state and local medical societies.

MARK WALTON JACOBY

Dr. Mark Walton Jacoby, who was born on March 7, 1893, died in Cleveland, Ohio, on March 28, 1957.

Dr. Jacoby received his degree as Doctor of Medicine from Ohio State University Medical School in 1917. He was chief of ophthalmology at St. Luke's Hospital in Cleveland at the time of his death.

A diplomate of the American Board of Ophthalmology, Dr. Jacoby was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the Association for Research in Ophthalmology, the Cleveland Academy of Medicine, the American Medical Association, and state and local medical societies. He was a past president of the Cleveland Ophthalmological Club.

Dr. Jacoby is survived by his wife and one son.

WALTER JELLIS

Dr. Walter Jellis was born in 1897 at Manchester, New Hampshire. He received his premedical education at Tufts College and his degree of Doctor of Medicine in 1920 from Tufts College Medical School. He did postgraduate work at New York Post Graduate Hospital and School in 1927 and 1928, after having a private practice in Somerville, Massachusetts since 1920.

Dr. Jellis, a specialist certified by the American Board of Otolaryngology, was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the Somerville Medical Society, the Massachusetts Medical Society, and the New England Otolaryngological Society. He had been a Lieutenant Commander of the United States Navy during World War II.

Dr. Jellis died on May 12, 1957. He is survived by one daughter, Mrs. William A. Sears, Needham, Massachusetts.

SIMON JESBERG

Dr. Simon Jesberg of Los Angeles, California died June 24, 1957, at the age of 70 years. He had been born in Franklin, Iowa, on September 16, 1886, and received his undergraduate education in McPherson, Kansas, and obtained his degree as Doctor of Medicine from the University of California Medical School in 1913. He interned at Los Angeles County General Hospital, and was a resident at Brooklyn Eye and Ear Hospital, specializing in otolaryngology.

Dr. Jesberg began private practice in Los Angeles in 1916. He founded the department of otolaryngology at the University of Southern California, and was professor emeritus of that university. He also founded the departments of otolaryngology at the Los Angeles County General Hospital in 1916, and at Eye and Ear Hospital in Los Angeles in 1922. He was co-founder of the Research Study Club of Los Angeles in 1922. During World War I, Dr. Jesberg served as a captain at Base Hospital No. 35 in France for two years.

Dr. Jesberg was the first otolaryngologist to perform a laryngectomy in California (1916). He presented to the University of Southern California Medical School a collection of 1500 foreign bodies removed from the food and air passages of some of his patients. He was the first person in Los Angeles to use radiation for treating cancer.

Besides his fellowship in the American Academy of Ophthalmology and Otolaryngology, Dr. Jesberg was a member and a former vice-president of the American Laryngological, Rhinological, and Otological Society, a member of the Los Angeles County Society of Ophthalmology and Otolaryngology, of the Pacific Coast Ophthalmologic and Otolaryngologic Society, of the Triological Society, of the American Broncho-Esophagological Society, of the American Medical Association, and of state and local medical societies.

Dr. Jesberg enjoyed many hobbies including fishing, hunting, sailing, music, and cooking.

Survivors include his wife, Ruth O. Jesberg, and sons, Dr. Norman Jesberg, who is also a member of the American Academy of Ophthalmology and Otolaryngology and Robert Jesberg, all of Los Angeles.

HARRY VINCENT JUDGE

Dr. Harry Vincent Judge, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died in Albany, New York, on March 3, 1957.

Dr. Judge was born on May 11, 1889, in Albany, and received his degree as Doctor of Medicine from Albany Medical College in 1914. After a residency in ophthalmology at Wills Eye Hospital in Philadelphia, he returned to his native city to engage in the practice of his specialty in 1918.

From 1927 until his retirement in 1954, Dr. Judge was professor of ophthalmology at Albany Medical College, and head of the department of ophthalmology at Albany Hospital. He was also on the staffs of Anthony M. Brady Maternity Home, St. Margaret's Hospital, and Greene County Memorial Hospital.

Dr. Judge was a diplomate of the American Board of Ophthalmology. He was a fellow of the American College of Surgeons and the New York Academy of Medicine, and held membership in the New York Ophthalmological Society, the Eastern New York Eye, Ear, Nose and Throat Association, the American Medical Association, and state and local medical societies.

Dr. Judge is survived by his wife, Cornelia Mills Judge, and one son, Dr. Harry M. Judge, also a Fellow of the Academy.

HENRY KAPP

Dr. Henry Kapp was born in Russia on September 3, 1889. When he was three years old his parents came to America and settled in Lawrence, Massachusetts, where he attended school. He was graduated with the degree of Doctor of Medicine from Tufts College Medical School in 1911, and did postgraduate work in otolaryngology at Boston Floating Hospital and Boston City Hospital before beginning his practice in Haverhill in 1915.

During World War I, Dr. Kapp served as an officer in the Medical Corps. On his discharge he returned to Haverhill where he was on the staff of the Haverhill Municipal Hospitals. Since its organization in 1952, he was chairman of the Hale Hospital board of trustees and a member of its staff.

tified a specialist in that field by the American Board of Otolaryngology in 1926.

Dr. Key became a staff surgeon at Seton Infirmary and at St. David's Hospital in Austin, Texas; he was the otolaryngologist and ophthalmologist at Students Health Service at the University of Texas.

At the time of his death, September 3, 1956, Dr. Key was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, receiving the Honor Key in 1952. He also was a Fellow of the American College of Surgeons, a member of the Travis County Medical Society, the Texas State Medical Society, and the Southern Medical Society.

LeROY TAYLOR KINCANNON

Dr. LeRoy Taylor Kincannon was born in Verona, Mississippi, on June 29, 1894. His undergraduate work was done at the University of Mississippi, and the University of Virginia Department of Medicine conferred upon him the degree of Doctor of Medicine in 1920. His postgraduate work in otolaryngology was done at Manhattan Eye, Ear and Throat Hospital in New York City.

All of Dr. Kincannon's professional life was spent in Birmingham, Alabama, where he was formerly on the faculty of the Medical College of Alabama. He was chief of staff of the Otolaryngology Department at Children's Hospital, and a member of the staff of the Baptist and St. Vincent's hospitals and of South Highland Infirmary. During World War II, he served in the Army Medical Corps in the European theater.

Dr. Kincannon was a specialist certified by the American Board of Otolaryngology. He was a Senior Fellow of the American Academy of Ophthalmology, and a member of the American Medical Association and of state and local medical societies.

Dr. Kincannon died in Birmingham on November 28, 1956. He is survived by his wife, and one son, LeRoy Taylor Kincannon, Jr.

CHARLES ROYAL KING

Dr. Charles Royal King, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died in Toledo, Ohio, on June 22, 1956.

Dr. Kapp was a specialist certified by the American Board of Otolaryngology. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Medical Association and of state and local medical societies.

Dr. Kapp was an ardent and enthusiastic worker in the field of medicine and in the civic affairs of Haverhill. He had been honored in recent years by the Haverhill chapter of B'nai B'rith and by receipt of the Hale Hospital Certificate of Merit. He was a member of the American Jewish Congress and a life-long member of the Zionist Organization of America.

Dr. Kapp died at his home in Haverhill on July 24, 1956. He is survived by his wife, Mabel Cohen Kapp, and three daughters, Mrs. Helen Orenstein of Methuen, Massachusetts, and Mrs. Janet Kopel and Miss Barbara Kapp of Haverhill.

PERCIVAL KAUFMAN

Dr. Percival Kaufman of New York City, a Junior Member of the American Academy of Ophthalmology and Otolaryngology, and a specialist certified by the American Board of Otolaryngology, died in New York City on March 3, 1957, at the age of 64.

Dr. Kaufman received his degree as Doctor of Medicine from New York University College of Medicine in 1915, and began his professional career as clinical assistant in otolaryngology at the New York Polyclinic Hospital the following year. He had also served as chief of the DeMilt Ear, Nose and Throat Clinic from 1917 to 1922.

Dr. Kaufman had spent his entire professional life in his native city, where he was on the staff of Midtown Hospital at the time of his death.

SAMUEL NEWTON KEY

Dr. Samuel N. Key was born in 1887 at Georgetown, Texas. He was graduated from the University of Texas in 1910 with the degree of Doctor of Medicine. Postgraduate training was completed at Eye and Ear Hospital, Brooklyn in 1912 and 1913. In 1914 Dr. Key limited his practice to otolaryngology and was cer-

Dr. King was born in Whitehouse, Ohio, on October 31, 1885. He attended the University of Toledo and received his degree as Doctor of Medicine from the medical department of his alma mater in 1907. He began the practice of medicine in his native city the same year, but within a few years he returned to Toledo where he became a specialist in ophthalmology and otolaryngology.

In addition to his fellowship in the American Academy of Ophthalmology and Otolaryngology, Dr. King was a fellow of the American College of Surgeons, and a member of the American Medical Association and of state and local medical societies.

GEORGE MADISON KRIEGER

Dr. George M. Krieger, who was born in 1881, died in Michigan City, Indiana, on May 24, 1956, at the age of 75 years.

In 1906 Dr. Krieger was graduated from Miami College in Cincinnati with the degree of Doctor of Medicine. He had limited his practice to the special fields of otolaryngology and ophthalmology since 1920.

Dr. Krieger was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Medical Association and of state and county medical societies.

NATHAN KENT LAZAR

Dr. Nathan Kent Lazar was born in Russia on March 1, 1897. He received his degree as Doctor of Medicine from the University of Illinois Medical School in 1927, and did postgraduate work in Prague the following year.

In 1929 Dr. Lazar began the practice of ophthalmology in Chicago, where he was assistant professor of ophthalmology at Northwestern University Medical School. He was attending ophthalmologist at Passavant Memorial Hospital.

Dr. Lazar was a diplomate of the American Board of Ophthalmology. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and of the American College of Surgeons. He also held membership in the Chicago Ophthalmological Society, in the American Medical Association, and in state and local medical societies.

Dr. Lazar died in Miami Beach, Florida, on December 14, 1956. He is survived by his wife, Marion; and one daughter, Barbara Lazar Wiesenfeld.

LAVEL HALL LEESON

Dr. Lavel H. Leeson was born August 17, 1890, in Vancouver, B. C., Canada. He received his premedical education in Vancouver schools, and his degree of Doctor of Medicine from McGill University in 1915. He was certified a specialist in ophthalmology and otolaryngology by the Royal College of Physicians and Surgeons of Canada.

Dr. Leeson died June 29, 1957. He had been a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the Pacific Coast Oto-Ophthalmological Society, the American Society of Ophthalmologic and Otolaryngologic Allergy, the American College of Surgeons, and the Canadian Otological Society. He had been a consultant at Children's Hospital, Vancouver, and also at Vancouver General Hospital. He was a professor at the University of British Columbia, in the eye, ear, nose, and throat section.

FORREST SHETTERLY LeTELLIER

Dr. Forrest Shetterly LeTellier was born in Knoxville, Tennessee, on August 25, 1893. He received his degree as Doctor of Medicine from the University of Tennessee College of Medicine in Memphis in 1917, and served in France as a first lieutenant in the Army Medical Corps during World War I.

In 1918, Dr. LeTellier returned to his native city to begin the practice of medicine. In 1922 he entered postgraduate study at the Chicago Eye, Ear, Nose and Throat Hospital and upon completion of his fellowship, he became certified by the American Board of Ophthalmology.

Dr. LeTellier continued the practice of his specialty in Knoxville until his death on October 15, 1956. He was a member of the staffs of St. Mary's Memorial, Fort Sanders Presbyterian, and Knoxville General hospitals.

Dr. LeTellier was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and of the American College of Surgeons. He was past president of the Tennessee Valley Postgraduate Medical Assembly, and a member of the American Medical Association and of state and local medical societies.

Dr. LeTellier is survived by his wife,

Emily Hall LeTellier; his stepson, Dr. John H. Saffold; and his mother, Lucy Shetterly LeTellier.

JAMES PRINCE LEWIS

Dr. James Prince Lewis, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, was born in Portland, Maine, in 1881. He was graduated from Harvard Medical School, Boston, in 1904 with the degree of Doctor of Medicine, and did postgraduate work in otolaryngology at Boston City Hospital, Rhode Island General Hospital, St. Elizabeth's Hospital, and Children's Hospital. He served on the faculty of Tufts College Medical School and was associated with the Waltham (Mass.) Hospital, Boston Floating Hospital, Cambridge City (Mass.) Hospital, and the Boston Dispensary.

Certified as a specialist by the American Board of Otolaryngology, Dr. Lewis was a member of the Waltham Medical Club, of the Northeast Oto-Laryngological Society, and of the American Medical Association, and state and local medical societies.

Dr. Lewis died in Boston on May 31, 1957.

JOSEPH DAVIDSON LEWIS

Dr. Joseph Davidson Lewis was born in 1871 at New Castle, Pennsylvania. He was graduated from Western Reserve Medical College in 1893 as a Doctor of Medicine. He interned at New York Ophthalmic and Aural Hospital, and began private practice in New York City. He later moved to St. Paul, Minnesota, where he was in private practice and was also Chief of the Department of Otolaryngology at Minneapolis General Hospital from 1913 to 1922. In 1922 Dr. Lewis moved to Santa Barbara, California, where he was on the staffs of Cottage Hospital and St. Frances Hospital. He was also Chief of the Department of Otolaryngology at Santa Barbara General Hospital.

In 1939 Dr. Lewis underwent a complete laryngectomy, but resumed his practice in Santa Barbara shortly thereafter. In 1946 he was operated upon for a cataract, and again underwent this operation in 1953. Dr. Lewis still practiced in Marysville, California, even though he had vision in only one eye. From 1945 to 1954, when he retired, he was practicing once more in Santa Barbara.

Dr. Lewis was the author of many medical papers and monographs, and was the inventor of several hospital necessities, including a dishwasher, sterilizers, sprinklers, gaskets, and other medical innovations.

When Dr. Lewis died on May 31, 1957, he was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, a member of the Santa Barbara Medical Society, California Medical Association, Los Angeles Society of Ophthalmology and Otolaryngology, American Medical Association, and Pacific Coast Oto-Laryngological Society. He also was President of the Tri-County Medical Society.

Survivors include Dr. Lewis's wife, Julie C. Lewis, one son, Fielding O. Lewis, retired ophthalmologist and also a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a daughter, Marcea Lewis Reynolds.

HAROLD IRVING LILLIE

Dr. Harold Irving Lillie, 69 years of age, former member of the staff of the Mayo Clinic for thirty-six years and renowned specialist in diseases of the ear, nose, and throat, died at his home in Rochester, Minnesota, on August 27, 1957, after a long illness.

Dr. Lillie was born May 6, 1888, in Grand Haven, Michigan. He received his degree of Bachelor of Arts in 1910 and the degree of Doctor of Medicine in 1912 from the University of Michigan. From 1912 to 1915 he was an intern in the University of Michigan Hospital, and from 1913 to 1915 was an instructor in otolaryngology in the University's medical school. In 1915 Dr. Lillie moved to Chicago where he practiced his specialty of otolaryngology and was an instructor at Rush Medical College. He moved to Rochester in 1917 and became Chief of the Section on Otolaryngology and Rhinology, a position which he held until becoming senior consultant on October 1, 1951. He retired from the Mayo Clinic on July 1, 1953.

Dr. Lillie was president of the American Rhinological, Laryngological and Otological Society in 1939 and of the American Laryngological Society in 1945

and 1946. He was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, a fellow of the American College of Surgeons, the International College of Surgeons, and a member of the Minnesota Academy of Ophthalmology and Otolaryngology, of the American Otological Society, and of the American Medical Association and state and local medical societies. For many years Dr. Lillie devoted much time to the breeding and raising of horses which were entered in many county fairs. He was a highly respected official at Olmsted County Fairs.

In 1913 Dr. Lillie married Oda Kittridge, who survives with a son, Dr. John C. Lillie, who is a member of the staff of the Mayo Clinic in the section which his father established and is also a Fellow of the American Academy of Ophthalmology and Otolaryngology, and one daughter, Mrs. Thomas C. Wilder of Spokane, Washington.

HUGH MONTGOMERY LOKEY

Dr. Hugh Montgomery Lokey, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, was born in Cherokee County, Alabama, on November 19, 1876.

Dr. Lokey received his degree in medicine from the Atlanta College of Physicians and Surgeons, now Emory University School of Medicine, in 1900. For two years following his graduation, he engaged in the general practice of medicine in Coosa, Georgia, but in 1902 he became associated with Dr. Abner W. Calhoun of Atlanta in the practice of medicine as related to the eye, ear, nose and throat, a practice which he continued throughout his professional life. He was on the staffs of Wesley Memorial and Piedmont hospitals in Atlanta and a consultant for the Georgia Baptist Hospital. For many years he was associated with the Good Samaritan Clinic and served on its Board of Trustees.

In addition to Life Fellowship in the Academy, Dr. Lokey was a fellow of the American College of Surgeons and a member of the American Medical Association. He was also a member of state and local medical societies. He was a past president of the Fulton County Medical Society.

Dr. Lokey died in Atlanta, on February 23, 1957, aged 80 years. He is survived by his wife, Rebecca Hamilton Lokey, and four children.

SAMUEL HERMAN LONG

Dr. Samuel Herman Long was born in Lithuania on October 22, 1885. His preliminary education was obtained in Evergreen, Alabama, and the University of Maryland School of Medicine conferred upon him the degree of Doctor of Medicine in 1909. Postgraduate work in otolaryngology was done at the University of Vienna.

In 1913 Dr. Long began the practice of his specialty in Chattanooga, Tennessee, and continued to practice there until his death on November 10, 1956. He was on the teaching staff of the Baroness Erlanger School of Nursing, and was a member of the staffs of the Baroness Erlanger, Memorial, Tri-County, and T. C. Thompson Children's hospitals.

A specialist certified by the American Board of Otolaryngology, Dr. Long was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and of the Tennessee Academy of Ophthalmology and Otolaryngology. He was a member of the American Medical Association and of state and local medical societies. He had served as president of the Chattanooga County Medical Society and of Hamilton County Medical Society.

Dr. Long is survived by his wife, Ruth Lachman Long; and one son, Ira M. Long, who is also a Fellow of the Academy.

JULIAN BARNETT MARKS

Dr. Julian Barnett Marks, a Senior Member of the American Academy of Ophthalmology and Otolaryngology, died in Los Angeles on April 5, 1957, at the age of 65 years. In 1918 he had received the degree of Doctor of Medicine from the University of Illinois College of Medicine, Chicago. He was a specialist certified by the American Board of Otolaryngology.

Dr. Marks was a member of the American Medical Association, and of state and local medical societies, and had served on the staffs of hospitals in the Los Angeles area.

ROY DAVID MARTIN

Dr. Roy David Martin, a Senior Member of the American Academy of Ophthalmology and Otolaryngology, was 72 years of age when he died at Grand Island, Nebraska, on July 1, 1957.

Dr. Martin had received his degree as Doctor of Medicine from the University of Nebraska, Omaha, in 1911. He was a specialist certified by the American Board of Otolaryngology, and had served on the staffs of the Grand Island Lutheran, and St. Francis hospitals. However, he had been retired for a number of years because of ill health.

JAMES ERNEST McASKILL

Dr. James Ernest McAskill was born in Highgate, Ontario, Canada, on January 31, 1888. He received his degree as Doctor of Medicine from Queen's University, Kingston, Ontario, in 1914, and served in the Canadian Army Medical Corps until the close of World War I, when he was discharged as major.

Following his discharge from the Army, Dr. McAskill entered a three-year residency in otolaryngology at Manhattan Eye and Ear Hospital. Upon its completion, he began the practice of his specialty in Watertown, New York, where he continued until his death on October 6, 1956. He had served on the staffs of Mercy Hospital and of Good Samaritan Hospital. He was a past president of the staff at Mercy Hospital and an honorary member of both staffs.

Dr. McAskill was a specialist certified by the American Board of Otolaryngology. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons, and he held membership in the American Laryngological, Rhinological and Otological Society, the American Medical Association and state and local medical societies. He had served as president, vice-president and treasurer of the Jefferson Medical Society, as president and secretary of the Fifth District Branch of the Medical Society of the State of New York, and as vice-president of the Medical Society of the State of New York.

Dr. McAskill was a past president of the Queen's University Alumni Association, and at the time of his death was a member of the university's board of trustees.

Surviving Dr. McAskill are his wife, Edith Stenton McAskill; and one son, James.

FREDERIC JOSEPH McINTIRE, JR.

Dr. Frederic Joseph McIntire, Jr. was born November 22, 1914, in Lynn, Massachusetts. He was graduated from Governor Dummer Academy and Dartmouth College and received his Doctor of Medicine degree from New York University College of Medicine in 1943. He then served his internship at Fordham Hospital and Bellevue Hospital, New York City. In March 1949, Dr. McIntire began private practice in Lynn and was on the staffs of Massachusetts Eye and Ear Infirmary, Lynn Hospital, and Union Hospital.

During World War II, Dr. McIntire was captured in the Battle of the Bulge and spent five months in a prison camp. He was known in Lynn as the "fighting doctor." He was a specialist certified by the American Board of Otolaryngology, a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Medical Association, of the New England Medical Society, of the Lynn Medical Society, and of the Essex South Medical Society.

Dr. McIntire died June 18, 1957, at Marblehead, Massachusetts. He is survived by his wife, Elizabeth Sheils McIntire, his parents, Dr. and Mrs. Frederic J. McIntire, Sr., and five children, Sheila, Maurie, Lizbeth, Frederic J., III, and Jean Marie.

OLNEY B. MONOSMITH

Dr. Olney B. Monosmith of Lorain, Ohio, received his medical degree from the College of Physicians and Surgeons of Chicago in 1893. He was a specialist certified by the American Board of Ophthalmology and served on the staff of St. Joseph Hospital in Lorain, where he died March 8, 1957, at the age of 85.

Dr. Monosmith was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, a fellow of the American College of Surgeons, and a member of state and local medical societies.

LEWIS FRANCIS MORRISON

Dr. Lewis Francis Morrison was born in San Francisco, California, on April 7, 1899. He attended the University of California, and the School of Medicine of his alma mater conferred upon him the degree of Doctor of Medicine in 1926. His training in otolaryngology was obtained at the University of California Medical School under the preceptor system.

Since 1926, Dr. Morrison engaged in the private practice of otorhinolaryngology in San Francisco, where he was a member of the faculty of otolaryngology at his alma mater. At the time of his death on November 10, 1956, he was clinical professor and chairman of the department. He was also visiting otolaryngologist at the University of California Hospital, University of California Out Patient Department, the Lagunda Honda Home, and San Francisco Hospital. He was consulting otolaryngologist at Infant's Shelter, and a consultant in otolaryngology for the Veterans Administration Hospital at Fort Miley and at Letterman General Hospital.

A specialist certified by the American Board of Otolaryngology, Dr. Morrison was an active member of the Board. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and had received its Honor Certificate. At the time of his death he was president-elect of the American Laryngological, Rhinological, and Otological Society, and a past president of the Pacific Coast Oto-Ophthalmological Society. Dr. Morrison held membership in the American Laryngological Association, the American Otological Society, the American Broncho - Esophagological Association, and was a fellow of the American College of Surgeons and of the Royal Society of Medicine of London, England. He was also a member of the American Medical Association and of state and local medical societies.

Always interested in teaching and advancing the specialty of otolaryngology, Dr. Morrison developed the residency training program at the University of California Medical School. He was the author of many scientific papers in the field of laryngology. He was particularly interested in bilateral abductor paralysis and the anatomy of recurrent laryngeal nerves. He was a dynamic, forceful worker continually striving for progress in the field of otolaryngology.

Dr. Morrison is survived by his wife, Dorothy Doyle Morrison; a daughter, Helen Morrison Elkus; and a son, William.

JOHN SANFORD MOYER

Dr. John S. Moyer, a Fellow of the American Academy of Ophthalmology and Otolaryngology, died July 20, 1956, at Metuchen, New Jersey. He was 60 years of age.

Dr. Moyer had received the degree of Bachelor of Science in 1919, and the degree of Doctor of Medicine in 1921 from the University of Pittsburgh. He completed postgraduate work in otolaryngology at the University of Pennsylvania in 1928, and had been associated with the Chevalier Jackson Bronchoscopic Clinic in Philadelphia. He had been assistant surgeon at Manhattan Eye, Ear, Nose, and Throat Hospital, New York City, assistant in the Allergy Clinic and Roosevelt Hospital, and served as medical officer of the Eye, Ear, Nose, and Throat Clinic (U. S. Public Health Service) at the United States Marine Hospital, at Ellis Island, N. Y.

At the time of Dr. Moyer's death, he was a fellow of the American College of Surgeons, and a member of the American Broncho-Esophagological Association, the New York State Medical Society, New York County Medical Society, and the American Medical Association.

EUGENE LEE MYERS

Dr. Eugene Lee Myers, a lifelong resident of St. Louis, was born in that city on May 27, 1880. He received his degree as Doctor of Medicine from St. Louis College of Physicians and Surgeons in 1909. In 1913 he went to Vienna for postgraduate study in otolaryngology, and in 1925 he studied broncho-esophagology under Chevalier Jackson at the University of Pennsylvania.

Dr. Myers was assistant professor of otolaryngology at St. Louis University School of Medicine, and had served on the consulting staff at Jewish Hospital for 32 years until his retirement about three years ago.

Dr. Myers was a diplomate of the American Board of Otolaryngology. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Medical Association and of state and local medical societies.

Dr. Myers died in St. Louis on March 12, 1957. He is survived by his wife and one daughter.

HARLEY DORE NEWBY

Dr. Harley Dore Newby was born in Parker, South Dakota, on November 12, 1885. He was graduated from the University of South Dakota and received his degree as Doctor of Medicine from Rush Medical College in 1911.

From 1911 until our entry into World War I, Dr. Newby engaged in the general practice of medicine in his native city. During the war he served in the Medical Corps. Upon his discharge he returned to Rush Medical College for specialty training in otolaryngology. After completion of his residency, he practiced his specialty in Rapid City, South Dakota, where he was on the staffs of St. John's, McNamara and Bennett Memorial hospitals.

A specialist certified by the American Board of Otolaryngology, Dr. Newby was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Medical Association and of state and local medical societies.

Dr. Newby died in Rapid City on November 1, 1956. He is survived by his wife and by two sons, who are both in the Air Force.

JOHN HENRY OHLY

Dr. John Henry Ohly was born in Brooklyn, New York, on September 10, 1877. He received his degree as Doctor of Medicine from Long Island College Hospital in Brooklyn, in 1899. After his internship he went abroad for postgraduate study in ophthalmology, studying under Fuchs in Vienna and Silex in Berlin. He also attended eye clinics in Halle and in London.

Returning to his native city after three years, he began the practice of ophthalmology which he continued until his death on December 13, 1956. He was active on the staffs of the Brooklyn Eye and Ear Hospital, Kings County Hospital, and Norwegian Hospital, subsequently becoming senior surgeon at the Eye and Ear Hospital, and consultant at Swedish, Long Island College, St. Peters and Caledonian hospitals.

A specialist certified by the American Board of Ophthalmology, Dr. Ohly was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons. He also held membership in the American Ophthalmological Society, in the Association for Research in Ophthalmology, in the Brooklyn Ophthalmological Society, and in the American Medical Association and state and local medical societies. He was a member of long standing and a past president of the New York Ophthalmological Society.

Dr. Ohly is survived by one son, John Henry, Jr.

RICHARD JOHNSON PAYNE

Dr. Richard Johnson Payne, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died in St. Louis, Missouri, on August 28, 1956.

Dr. Payne was born April 14, 1888. He received the degree of Doctor of Medicine from St. Louis University School of Medicine in 1913. His postgraduate work in otolaryngology was done at the University of Pennsylvania.

The entire professional life of Dr. Payne had been passed in St. Louis, Missouri. He served on the faculty of Washington University Medical School, and had been a member of the staffs of Barnes, Childrens, Missouri Baptist, St. Mary's, Faith, and Frisco hospitals.

Dr. Payne was a specialist certified by the American Board of Otolaryngology. In addition to his fellowship in the American Academy of Ophthalmology and Otolaryngology, Dr. Payne was a member of the American Medical Association and of state and local medical societies.

Dr. Payne is survived by his wife, Mary Mathews Payne; and two sons, W. Spencer and Richard, Jr.

LOUIS M. PEARLMAN

Dr. Louis M. Pearlman was born Feb-

ruary 14, 1888. He was graduated in 1911 from the Long Island College Hospital, New York.

Dr. Pearlman was a Junior Fellow of the American Academy of Ophthalmology and Otolaryngology, and was a specialist certified by the American Board of Otolaryngology. He was a professor at Polyclinic Hospital and a member of the New York County Medical Society, New York State Medical Association, and American Medical Association, at the time of his death in 1953.

FRANCIS PETER QUINN

Dr. Francis Peter Quinn was born in Pomeroy, Iowa, on April 15, 1893. He was graduated from the State University of Iowa and his alma mater conferred upon him the degree of Doctor of Medicine in 1921. He entered a residency in the same institution and received his Master of Science degree in otolaryngology in 1923.

Dr. Quinn began the practice of his specialty in Dubuque, Iowa, in 1923, and remained there until his death on August 8, 1956.

Dr. Quinn was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Medical Association, of the Mississippi Valley Medical Society, and of state and local medical societies.

Dr. Quinn is survived by his wife and two sons.

GEORGE F. REINEKE

Dr. George F. Reineke, aged 76 years, died at his home in New Ulm, Minnesota on May 21, 1957. He had received his degree as Doctor of Medicine from the Medical School of the University of Minnesota, had been certified a specialist by the American Board of Otolaryngology, and had served on the staffs of the Loretto and the Union Hospitals in New Ulm for many years.

Dr. Reineke considered it a great privilege to remain a member of the American Academy of Ophthalmology and Otolaryngology even though he was unable to attend meetings or take an active part in its activities, due to cataracts which caused partial blindness. He was also a member of the Redwood-Brown County Medical Society, the State of Minnesota Medical Society, and the Sioux Valley Eye and Ear Academy.

GEORGE BOND RIDOUT

Dr. George Bond Ridout was born in New Jersey on January 4, 1901. He was a graduate of Syracuse University and received his degree as Doctor of Medicine from the University of Louisville School of Medicine in 1926. His postgraduate work in otolaryngology was done at the Graduate School of the University of Pennsylvania. In 1934 he took a basic course in Ophthalmology at the New York Eye and Ear Infirmary, and in 1935 he entered a residency in ophthalmology and otolaryngology at the Naval Hospital in Washington.

From 1928 to 1945, Dr. Ridout served in the Medical Corps of the United States Navy. Following his separation from the Navy, he became affiliated with the Gundersen Clinic at LaCrosse, Wisconsin, where he remained until his death on November 26, 1956. He also served on the staff of the Lutheran Hospital in LaCrosse.

A diplomate both of the American Board of Ophthalmology and of the American Board of Otolaryngology, Dr. Ridout was a Fellow of the American Academy of Ophthalmology and Otolaryngology. He was also a member of the American Association of Railway Surgeons, of the American Medical Association, and of state and local medical societies.

Dr. Ridout is survived by one daughter, Elizabeth, who lives in Washington, D. C.

OTTO CARL RISCH

Dr. Otto Carl Risch was born in Syracuse, New York, on June 21, 1893. He attended Columbia University and Syracuse University, and was granted the degree of Doctor of Medicine by the Syracuse University College of Medicine in 1920. His postgraduate work in otolaryngology was done at Manhattan Eye and Ear Hospital in New York City.

Dr. Risch began the practice of general medicine in Passaic, New Jersey, but returned to New York City in 1926, where he limited his practice to otolaryngology. He was director of otolaryngology at French Hospital in New York City and at the Municipal Sanitorium at Otisville, New York. He was a consultant in otolaryngology at Flushing Hospital and Queens Speech and Hearing Center in Flushing, New York,

and at Lutheran Hospital in Brooklyn. He also served on the courtesy staff at St. Clare's and Midtown hospitals in New York City.

A specialist certified by the American Board of Otolaryngology, Dr. Risch was a Fellow of the American Academy of Ophthalmology and Otolaryngology, of the American College of Surgeons, of the American Academy of Chest Physicians, and of the New York Academy of Medicine. He also held membership in the American Laryngological, Rhinological and Otological Society, the American Otological Society, the American Medical Association and state and local medical societies. He served as secretary for the Section of Otolaryngology for the New York Academy of Medicine and for the Committee of Consultants on Occupational Loss of Hearing, as well as for the New York State Workman's Compensation Board. He was a past president of the Medical Board of New York City Hospital and of Municipal Sanitorium in Otisville, New York; and a member of the Board of Governors of the American Academy of Compensation Medicine.

Dr. Risch died in Flushing, Long Island, on November 11, 1956. He is survived by his wife and one son.

EDWARD DANIEL RISSER

Dr. Edward D. Risser of Pasadena, California, died May 1, 1957, at the age of 66 from coronary thrombosis. In 1918 he was graduated from the State University of Iowa College of Medicine, Iowa City, Iowa. He was a specialist certified by the American Board of Otolaryngology and practiced in Winona, Minnesota, for several years. After he moved to California he was associated with St. Luke, Alta Vista, and the Collis P. and Howard Huntington Memorial hospitals in Pasadena.

Dr. Risser was a Fellow of the American Academy of Ophthalmology and Otolaryngology and was at one time vice-president of the Minnesota Academy of Ophthalmology and Otolaryngology.

KERSEY CLYDE RITER

Dr. Kersey Clyde Riter was born in Logan, Utah, on December 8, 1906. He received his degree as Doctor of Medicine from Northwestern University School of Medicine in 1937. From 1938 until 1941, he did postgraduate work at New York Postgraduate Hospital, completing his work in otolaryngology in 1940 and his work in ophthalmology in 1941.

His first practice at the Budge Clinic in Logan, Utah, was interrupted by military duty in the Medical Corps in September 1942. Upon his discharge from the Medical Corps in January 1946, Dr. Riter returned to Logan, where he was on the staff of the Latter-Day Saints Hospital. In 1950, Dr. Riter returned to Salt Lake City, where he continued practice until his death on July 23, 1956.

A specialist certified by the American Board of Otolaryngology, Dr. Riter was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons. He also held membership in the Pan-American Association of Ophthalmology, the Intermountain Oto-Ophthalmological Society, the American Medical Association and state and local medical societies.

Dr. Riter is survived by one son, Samuel, who is now serving in the United States Army.

DANIEL ABELL RUSSELL

Dr. Daniel Abell Russell was born October 22, 1895, in San Antonio, Texas. He was a graduate of the University of Texas and received his degree as Doctor of Medicine from Tulane University School of Medicine in 1921.

In 1922 he began the practice of medicine in San Antonio, Texas, where he served as chief of otolaryngology at Santa Rosa Hospital, and as consultant in otolaryngology at Brooke Army Medical Center, Robert B. Green Hospital, and Nix Hospital.

Dr. Russell was a specialist certified by the American Board of Otolaryngology. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the Texas Society of Ophthalmology and Otolaryngology, the San Antonio Society of Ophthalmology and Otolaryngology, the American Medical Association and state and local medical societies.

Dr. Russell died in his native city on February 2, 1957. He is survived by his wife, Naomi Gouger Russell; by two daughters, Mrs. James Folbre and Mrs. Michael Kangerga; and by one son, Daniel A. Russell, who is also a Fellow of the Academy.

ALBERT EUGENE SELIGMAN

Dr. Albert Eugene Seligman was born March 4, 1891, in Frankfurt on the Main, Germany. His education was obtained at Frankfurt on the Main Gymnasium and the degree of Doctor of Medicine was conferred upon him by the University of Heidelberg Medizinische Fakultat, Baden, Germany, in 1916. He began practicing his specialty in Germany in 1920. In the United States Dr. Seligman was on the staffs of the Willowbrook State School, the Richmond Memorial Hospital, Staten Island Hospital, St. Vincent's Hospital, Willowbrook Hospital, and Sunnyside Hospital.

Dr. Seligman was a diplomate of the American Board of Otolaryngology. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the Richmond County Medical Society, of the New York State Medical Society, and of the American Medical Association.

On June 17, 1957, Dr. Seligman died of leukemia at his home in Staten Island. He is survived by his wife, a daughter, Marianne of Cantor, New York, and a son, Walter.

EDWARD CECIL SEWALL

Dr. Edward Cecil Sewall was born June 15, 1875, in Portland, Oregon. His preliminary education was obtained at Leland Stanford University, Palo Alto, California. In 1902 he received his degree as Doctor of Medicine from Cooper Medical College, San Francisco. He then became associated with Dr. Adolph Barkan, and also began a teaching position at Cooper Medical College. He completed his postgraduate study in Vienna, London, and Paris.

In 1912 Cooper Medical College became the Medical School of Stanford University, at which time Dr. Sewall became Clinical Professor and Chief of the Eye, Ear, Nose, and Throat Section. In 1914 he discontinued all eye work and continued as Chief of Otolaryngology until his retirement from the University in 1940.

Dr. Sewall was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Medical Association, of the Pacific Coast Eye, Ear, Nose, and Throat Society, and of the American Laryngological Association. He was an honorary member of the Dallas Southern Clinical Society.

On April 9, 1957, Dr. Sewall died in Palo Alto at the age of 82 years. He is survived by his wife, the former Amy Heitshu.

FRANCIS XAVIER SIEGEL

Dr. Francis Xavier Siegel, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died April 10, 1957, at St. Francis Hospital in Cincinnati at the age of 75.

Dr. Siegel was born in Springfield, Ohio, on October 12, 1881. He received his undergraduate education at St. Bernard Parochial School, Springfield High School, and Wittenberg College, and his premedical training at St. Xavier's College. He received his Doctor of Medicine degree from the Miami Medical College of the University of Cincinnati in 1907. He established a private practice in 1908 in Cincinnati, where he served on the staffs of St. Mary's Hospital, the Good Samaritan Hospital, St. Francis Hospital, and Deaconess Hospital.

Certified as a specialist by the American Board of Ophthalmology, Dr. Siegel was a fellow of the American College of Surgeons and of the International College of Surgeons. He also held membership in the Cincinnati Academy of Medicine, in the American Medical Association, and in state and local medical societies.

He was credited with being the first doctor in Cincinnati to use gas as an anesthetic for a major operation, prior to World War I, and was one of the first surgeons in Cincinnati to perform the delicate keratoplasty operation.

His wife, Lillian Brigel Siegel, and one daughter, Mrs. Angelo Pilotto, wife of the internationally known baritone, survive.

MORRIS BIGELOW SIMPSON

Dr. Morris Bigelow Simpson was born in Buffalo, Kansas, on December 29, 1886. He was a graduate of Baker University in Baldwin, Kansas, and received his degree as Doctor of Medicine from the University of Kansas City Medical College in 1913. His first practice in Jew-

ell, Kansas, was interrupted by service as captain in the British Medical Corps in World War I. For this service he was awarded the British Military Cross and the French Croix de Guerre.

At the close of the war, Dr. Simpson entered a residency in otolaryngology at New York Eye and Ear Hospital, and completed his postgraduate work at the New York Polyclinic Hospital. He began the practice of his specialty in Kansas City, where he served on the staffs of St. Mary's, Children's Mercy, Kansas City General, and Research hospitals and the Menorah Medical Center.

A specialist certified by the American Board of Otolaryngology, Dr. Simpson was a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology. He also held membership in the Pan-American Ophthalmological Society and the World Medical Association. He had served as president of the Missouri State Medical Association (1947), the Kansas City Eye, Ear, Nose and Throat Society (1946), the Jackson County Medical Society (1937), and the Kansas City Southwest Clinical Society (1949).

Dr. Simpson died at his home, Morrelen Farm, at Kearny, Missouri, on November 10, 1956. He is survived by his wife.

FERRIS N. SMITH

Dr. Ferris N. Smith, a Fellow of the American Academy of Ophthalmology and Otolaryngology, died September 20, 1957, at Pontiac, Michigan, after an outstanding medical record in World War I and successful practices in Pontiac and Grand Rapids. He had taught plastic surgery in European hospitals and was the author of two textbooks, "Reconstructive Surgery" and "Plastic and Reconstructive Surgery," and monographs on reconstructive surgery and chronic sinus diseases, and was co-author of the army medical corps "Manual of Plastic and Maxillo-Facial Surgery."

Dr. Smith was born in Pontiac on October 25, 1884. He studied at the University of Michigan, where he received his Bachelor of Arts and Doctor of Medicine degrees, and then took postgraduate work in Vienna and in Berlin. He practiced medicine in Ann Arbor, Michigan, for some time and then began private practice in Pontiac in 1913. In World War I he was a captain in the English Royal Medical Corps and was one of two plastic surgeons at Queens Hospital, London. He then was professor of plastic surgery at the International Clinic in Paris and demonstrated at the Royal College of Surgeons in England. When he came to Grand Rapids in 1923, he joined the Blodgett Memorial Hospital staff and also served on the staffs of Butterworth and St. Mary's hospitals.

Dr. Smith was an honorary fellow of the International College of Dentists, and was affiliated with the American College of Surgeons, the Founders group of the American Board of Plastic Surgery, the American Association of Plastic Surgeons, and the American Society of Plastic and Reconstructive Surgery. He was past president of the Oral and Plastic Surgery Society.

FRANK ROBERT SPENCER

Dr. Frank R. Spencer was born June 12, 1879 at Burlington, Iowa. He obtained his early education in Burlington Public Schools. He received degrees of Bachelor of Arts and Doctor of Medicine from the University of Michigan and then spent two years in postgraduate work in the University's Department of Anatomy. He was temporary assistant at Manhattan Eye, Ear, Nose, and Throat Hospital for one year, and was also at the Massachusetts Eye and Ear Infirmary for six months. Six months were spent in Vienna before Dr. Spencer began private practice in Boulder, Colorado where he also was head of the Ear, Nose, and Throat Department of the University of Colorado until his retirement in 1947.

Dr. Spencer was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, a member of the American Medical Association, the American Laryngology, Rhinology, and Otology Association, the American Laryngological Society, and the American Otological Society.

Survivors are two sons; Dr. Robert Spencer of Denver, and Donald Spencer, a mathematician at Princeton University.

ABRAHAM BENNETT SPIEGELGLASS

Dr. Abraham B. Spiegelglass, aged 70 years, died November 13, 1957, in River Edge, New Jersey. He was a graduate of

the University of Pittsburgh School of Medicine in 1908 and was a specialist certified by the American Board of Otolaryngology.

Dr. Spiegelglass was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the Association for Research in Ophthalmology, and an associate member of the American Medical Association, a consultant at the Bergen Pines County Hospital in Paramus, and was on the staff of the Hackensack Hospital in Hackensack, and the Holy Name Hospital in Teaneck.

WILLIAM FRANCIS CHARLES STEINBUGLER

Dr. William F. C. Steinbugler, who had been born in New York City on June 2, 1886, died on April 7, 1957, in Garden City, New York.

Dr. Steinbugler had been graduated from Cornell University Medical College, New York City, in 1908 with the degree of Doctor of Medicine. His postgraduate work was done at Lenox Hill Hospital, Lying-in-Hospital, and Herman Knapp Memorial Eye Hospital. He began the practice of medicine in Brooklyn in 1912, and after 1917 his practice was limited to ophthalmology. He served on the staffs of Brooklyn Eye and Ear Hospital, Caledonian Hospital, Midwood Hospital, Harbor Hospital, and Mary Immaculate Hospital, all in Brooklyn.

Dr. Steinbugler was very active in church and medical affairs. He was one of the founders and also a president of the Brooklyn Ophthalmological Society. He was a welcome addition to any group because of his cheery and interested manner and always contributed something to any meeting or project.

Certified as a specialist by the American Board of Ophthalmology, Dr. Steinbugler was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons, and of the New York Academy of Medicine. He also was a member of the New York Ophthalmological Society, of which he was a past president, the Brooklyn Ophthalmological Society, and of the American Medical Association and of state and local medical societies. He also was co-founder and past president of the Catholic Physicians Guild of Brooklyn.

Mrs. Julia Dannahe Steinbugler survives with one daughter, Mrs. Edward (Claire) Moran, and twin sons, Richard (Frater M. Paul, o.c.s.o.) Trappist Monk in Lafayette, Oregon, and Thomas B., a Jesuit Scholastic, now teaching in the Ateneo de Manila, Manila, P. I.

WILLIAM HERMAN STOKES

Dr. William Herman Stokes, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died in Lake City, Michigan, on April 8, 1957.

Dr. Stokes was born in British East India on July 17, 1894. He was a graduate of Tubingen Gymnasium in Germany, coming to the United States in 1913. He was naturalized in 1918 when he was serving as a Lieutenant in the Sanitary Corps of the United States Army American Expeditionary Forces. He received his degree as Doctor of Medicine from the University of Michigan Medical School in 1922 and served a three year residency in ophthalmology at the same institution.

In 1925 Dr. Stokes became ophthalmologist for the Dallas Medical and Surgical Clinic in Dallas, Texas, where he remained in practice until he was appointed associate professor of ophthalmology at the University of Nebraska Medical College in 1930. In 1943 he was forced to resign as professor and chairman of the Department of Ophthalmology because of illness. Since his retirement, he had conducted a limited practice in Lake City, Michigan.

A specialist certified by the American Board of Ophthalmology, Dr. Stokes was a Fellow of the American College of Surgeons, and held membership in the American Ophthalmological Society, in the International College of Surgeons, in the American Medical Association and in state and local medical societies.

Dr. Stokes' death was a serious blow to the community where he had been serving and to his colleagues in Michigan. His frequent visits to the University of Michigan were always much appreciated by the ophthalmology staff because of the advice and counsel given the younger members.

Dr. Stokes is survived by his wife, Margaret Ann.

GILBERT STEVENS TABOR

Dr. Gilbert S. Tabor was born on August 17, 1898. He graduated from the University and Bellevue Hospital Medical College, New York City in 1919, and was a specialist certified by the American Board of Otolaryngology. He was past president of the Duchess County Medical Society, a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Oto-Rhinologic Society for Plastic Surgery, of the American Medical Association, and of state and local medical societies.

Dr. Tabor served as village health officer of Millerton, New York, and was a director of the Millerton National Bank. He was on the staff of the Sharon Hospital, Sharon, Connecticut.

Dr. Tabor died April 4, 1957, in Millerton.

GEORGE HOLTEN TRAUGH

Dr. George Holten Traugh was born in Ervona, Pennsylvania, on July 23, 1890. He received his degree as Doctor of Medicine from the University of Pittsburgh Medical School in 1919 and served for a short time as resident physician at McKeesport Hospital, McKeesport, Pennsylvania. He began the practice of medicine in Fairmont, West Virginia, as an associate of his uncle, Dr. John P. Trach, in 1923. In 1928 Dr. Traugh took postgraduate work in otolaryngology in Chicago and New York, and since that time had confined his practice to this specialty. He was on the staff of Fairmont General Hospital.

A specialist certified by the American Board of Otolaryngology, Dr. Traugh was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons. He was a member of Pittsburgh Otological Society, of the American Medical Association and of state and local medical societies.

Dr. Traugh died in Fairmont, on September 19, 1956. He is survived by his wife, Jean Picardo Traugh; and two sons, Walter C., and George Holten, Jr.

HARRY ALLEN VAN OSDOL

Dr. Harry Allen Van Osdol, who was born June 24, 1880, at Rising Sun, Indiana, died at his farm near Carmel, Indiana, on April 10, 1957.

Dr. Van Osdol received his degree as Doctor of Medicine from Indiana University School of Medicine in 1909, and did postgraduate work in otolaryngology at Johns Hopkins Hospital in Baltimore and at the Mayo Foundation of the University of Minnesota Medical School in Rochester, Minnesota.

Dr. Van Osdol had served on the staffs of St. Vincent's, Methodist, and Indianapolis General hospitals in Indianapolis. He was professor emeritus of otolaryngology at Indiana University School of Medicine.

Dr. Van Osdol was a diplomate of the American Board of Otolaryngology. He was a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology, and a Fellow of the American College of Surgeons. He was a past president of the Indianapolis Ophthalmological and Otolaryngological Society and of the Indiana Academy of Ophthalmology and Otolaryngology, and held membership in the American Medical Association and in state and local medical societies.

Dr. Van Osdol served as Captain in the Medical Corps of the United States Army during World War I. He was an enthusiastic farmer and a breeder of fine Percheron horses.

CLARENCE ARCHIBALD VEASEY

Dr. Clarence A. Veasey, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died August 11, 1957, at his home in Spokane, Washington, just two days after his eighty-eighth birthday.

Dr. Veasey was born in Pocomoke City, Maryland. He studied at Western Maryland College, and in 1890 received the degree of Doctor of Medicine from Jefferson Medical College in Philadelphia. Postgraduate work was done in European hospitals. From 1891 until 1908 Dr. Veasey engaged in the private practice of ophthalmology in Philadelphia, where he was also Clinical Assistant at Wills Eye Hospital for one year; then successively served as demonstrator, clinical assistant in ophthalmology, assistant professor of ophthalmology, and instructor in operative ophthalmology at

Jefferson Medical College until 1895. For the next five years Dr. Veasey was instructor of Operative Ophthalmology and Assistant Professor of Diseases of the Eye at the Philadelphia Polyclinic. He moved to Spokane, Washington in 1908, where he became associated with Deaconess and St. Luke's hospitals.

Certified by both the American Board of Ophthalmology and the American Board of Otolaryngology, Dr. Veasey was a member of the American Ophthalmological Society, of the Pacific Coast Oto-Ophthalmological Society, of the Spokane Academy of Ophthalmology and Otolaryngology, and of the American Medical Association and state and local medical societies. He was a fellow of the College of Physicians, Philadelphia, of the American Academy of Medicine, and of the American College of Surgeons. He was the author of "Ophthalmological Operations as Practiced on Animals," of a textbook on ophthalmology, and of numerous medical monographs.

Survivors are Dr. Veasey's wife, Gertrude M. Veasey, a son, Dr. Clarence A. Veasey, Jr., also a Fellow of the American Academy of Ophthalmology and Otolaryngology, and one daughter, Winona G. Veasey, all of Spokane.

JAMES WASHINGTON WARD

Dr. James Washington Ward, who was born in Bagwell, Texas, on August 8, 1879, died in Greenville, Texas, on December 16, 1956.

Dr. Ward received his degree as Doctor of Medicine from the University of Louisville in 1904. From 1904 until 1914 he conducted a general practice at Detroit, Texas, making calls in a buggy and on horseback. In 1914, he went to Manhattan Eye and Ear Hospital and the New York Eye and Ear Infirmary for postgraduate study in otolaryngology, and continued this study at the University of Vienna. From 1915 until 1955, when he retired, he limited his practice to the specialties of ophthalmology and otolaryngology.

Dr. Ward was a specialist certified by the American Board of Otolaryngology in 1926. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the Texas Society of Ophthalmology and Otolaryngology, the Dallas County Eye, Ear, Nose and Throat Society, the American Medical Association and state and local medical societies.

Dr. Ward is survived by his wife, two sons, and one daughter.

JOSEPH ROOSEVELT WEINTROB

Dr. Joseph Roosevelt Weintrob was born in Plainfield, New Jersey, on June 14, 1912. He was a graduate of Rutgers University and received his degree as Doctor of Medicine from Jefferson Medical College of Philadelphia, in 1937. His postgraduate work was done at the University of Pennsylvania Graduate School of Medicine.

Dr. Weintrob's entire professional life had been spent in Atlantic City, where he was chief of the Eye Service at Atlantic City Hospital.

A specialist certified by the American Board of Ophthalmology, Dr. Weintrob was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the Association for Research in Ophthalmology, of the New Jersey Academy of Ophthalmology and Otolaryngology, of the American Medical Association and of state and local medical societies.

Dr. Weintrob died in Atlantic City on January 28, 1957. He is survived by his wife, Beverly, and two small children.

PETER DE TILIUS WESTERHOFF

Dr. Peter De T. Westerhoff, 60 years of age, died suddenly June 22, 1957, at his home in Midland Park, New Jersey.

Dr. Westerhoff was born in Paterson, New Jersey, on December 29, 1896. He attended Ridgewood High School, Cornell and Wake Forest Universities, and Long Island Medical College. He interned at St. Joseph Hospital, Paterson, and began private practice in 1925. For nearly twenty-nine years he practiced his specialty in Midland Park. He was a staff member of St. Joseph Hospital, Paterson and was formerly associated with Columbia University and with the clinical staff of the Presbyterian Hospital in New York.

Dr. Westerhoff, a diplomate of the American Board of Otolaryngology, was a Fellow of the American Academy of Ophthalmology and Otolaryngology. He was also a member of the Passaic Coun-

ty Medical Society, the Medical Society of New Jersey, and the American Medical Association.

Survivors are his wife, Ruth Shaw Westerhoff; a son, Dr. Peter De T. Westerhoff, Jr., of Midland Park, also a Fellow of the American Academy of Ophthalmology and Otolaryngology; and three daughters, Mrs. Betty Artsma of Wyckoff, Mrs. Ruth Ryan of Ridgeport, Pennsylvania, and Barbara of Paterson.

MINOR ELISHA WHITE

Dr. Minor Elisha White was born in Dressor, Fayette County, Illinois, on November 18, 1889. He received his degree as Doctor of Medicine at Loyola University School of Medicine in Chicago in 1918. In 1924 he entered a three year residency in otolaryngology at the Illinois Eye and Ear Infirmary in Chicago, where he also served as attending physician and surgeon for two years.

In 1927 he moved to Kankakee, Illinois, where he was on the staff of St. Mary's Hospital and was local oculist and aurist for the Illinois Central Railroad. During World War II he served as chief of the otolaryngology department at Gorgas Hospital in the Canal Zone with the rank of colonel in the Army of the United States.

A specialist certified by the American Board of Otolaryngology, Dr. White was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the Pan-American Ophthalmological Society, of the American Medical Association and of state and local medical societies.

Dr. White died in Kankakee on December 26, 1956. He is survived by his wife, Jessie; a son, and two daughters.

CHARLES B. WYLIE

Dr. Charles B. Wylie, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died in Columbus, Ohio, on May 9, 1957, at the age of 93, from injuries received in a fall.

Dr. Wylie was graduated from Hering Medical College, Chicago, in 1903 with the degree of Doctor of Medicine, and was a specialist certified by the American Board of Otolaryngology.

Dr. Wylie was a resident of Morgantown, West Virginia, where he had been in private practice. He was an honorary member of the staffs of Monongalia General and Vincent Pallotti hospitals in Morgantown.

HERMAN EUGENE YAZEL

Dr. Herman E. Yazel of Kansas City, Missouri, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died at the age of 76 years, on January 19, 1957.

Dr. Yazel had been a graduate of the University of Missouri Medical College in 1907. He was a member of the American Medical Association and of state and local medical societies.

ARTHUR MEYER YUDKIN

Dr. Arthur Meyer Yudkin was born in Ansonia, Connecticut, on December 21, 1892. His undergraduate education was obtained at the Yale Sheffield Scientific School in New Haven, Connecticut, and he received his degree as Doctor of Medicine from Yale University School of Medicine in New Haven in 1917.

After serving his internship at New Haven Hospital, be became a clinical assistant in ophthalmology and otolaryngology in the Yale University School of Medicine. His entire professional career had been spent in the Department of Ophthalmology of his alma mater. At the time of his death on May 2, 1957, he held the position of clinical professor in ophthalmology. He also served as attending surgeon in ophthalmology at Grace-New Haven Hospital, as attending ophthalmologist at St. Raphael's Hospital, and as consulting ophthalmologist at Griffin Hospital.

Dr. Yudkin was a diplomate of the American Board of Ophthalmology. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Ophthalmologic Society, the Society for Experimental Biology and Medicine, the Society of Clinical Ophthalmology, the New York Academy of Medicine and the National Society for the Prevention of Blindness. He had served as co-editor of Practitioners Library of Medicine and Surgery.

Dr. Yudkin is survived by his wife, Adele Weissman Yudkin, and two sons, Marvin Yudkin and Dr. Gerald Yudkin.

BOOK REVIEW

ATLAS OF OTOLARYNGIC PATHOLOGY. By Col. J. E. Ash and Muriel Raum. Published jointly by the American Academy of Ophthalmology and Otolaryngology, American Registry of Pathology and the A.F.I.P., 1956. 572 pp. $20.00.

This book is a magnificent contribution to the otolaryngologic and general medical literature. It is a book of extreme importance not only to those interested in the pathology of otolaryngology, but also to clinical otolaryngologists and to general pathologists as well. The whole field is covered very nicely in both text and illustrations. The illustrations, which are profuse in number with accompanying legends, are outstanding both in their clarity and detail.

For the clinician and "occasional pathologist" there is a very nice review of general pathology, as it relates to the field of otolaryngology, occupying the first 109 pages. The remainder of the book is devoted to pathology and histopathology of the upper respiratory tract, the lower respiratory tract, the neck, and the ear. These sections contain a great deal of clinical information both in the text and in the illustration legends to orient the clinician. The illustrations are sufficient in number and detail to allow the clinician to classify material he might have on hand pretty much by comparison with the illustrations. At the same time the otolaryngic pathology material is presented in sufficient detail to be of interest and great value to the full time pathologist as a reference work.

In summary, the reviewer finds it difficult to express adequately the tremendous value and importance of this book. It is undoubtedly the most magnificent collection of clinical and histologic-pathological material ever gathered between two covers. It is a monumental work which should be on the desk of every otolaryngologist and every pathologist.

G. F. R.

News - - - Notes

A.M.A. SECTION ON OPHTHALMOLOGY ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY

CALL FOR PAPERS

The 1958 Meeting of the Section on Ophthalmology will be held in San Francisco, June 23-27, 1958, simultaneously with that of the Association for Research in Ophthalmology.

Anyone wishing to present a paper before the Section is urged to communicate with Dr. Harold G. Scheie, Secretary, Section on Ophthalmology, 313 South 17th Street, Philadelphia 3, Pennsylvania, as soon as possible. A title and abstract of 50 to 150 words must be in his hands by January 1, 1958. Anyone wishing to present a paper before the Association for Research should contact Dr. Lorand V. Johnson, Secretary, Association for Research in Ophthalmology, 10515 Carnegie Avenue, Cleveland 6, Ohio, as soon as possible.

A $250.00 prize is offered for the best paper presented before the Section at each Scientific Session.

CALL FOR EXHIBITS

The 1958 Meeting of the Section on Ophthalmology will be held in San Francisco, June 23-27, 1958, simultaneously with that of the Association for Research in Ophthalmology.

Anyone wishing to prepare a scientific exhibit should communicate with Dr. Frank W. Newell, 950 East 59th Street, Chicago 37, Illinois, as soon as possible.

A $250.00 prize is offered for the best scientific exhibit prepared for the Section on Ophthalmology at each Scientific Session.

NATIONAL COUNCIL TO COMBAT BLINDNESS

The Scientific Advisory Committee of the National Council to Combat Blindness, Inc., is now accepting applications for its 1958-59 FIGHT FOR SIGHT Grants-in-Aid, Research Fellowships and Summer (1958) Student Fellowships. The Committee will consider all applications at its Ninth Annual Meeting, scheduled in June 1958.

Completed applications for grants and fellowships must be in by March 1, 1958, in order to allow ample time for processing and review. Applications for Summer Student Fellowships will be reviewed in advance of the June meeting.

For the appropriate form, write to the Council's new address: Secretary, National Council to Combat Blindness, Inc., 41 West 57th St., New York 19, N. Y.

NATIONAL INSTITUTE OF NEUROLOGICAL DISEASES AND BLINDNESS

The Public Health Service is sponsoring a new program to give financial aid to research scientists who wish further training for research or teaching. The awards, made for not less than nine months and no more than one year, encourage training in neurochemistry, neuropharmacology, neurophysiology and neuroanatomy. All awards are subject to renewal.

Further information and application forms may be obtained by writing to the Chief, Extramural Programs Branch, National Institute of Neurological Diseases and Blindness, National Institutes of Health, Bethesda 14, Md.

CALIFORNIA

The American Laryngological, Rhinological and Otological Society, Western Section, will hold its Annual Meeting at the Beverly Hilton Hotel, Beverly Hills, January 18 and the morning of January 19, 1958. Members can then register for the meeting of the Research Study Club during the afternoon of January 19.

The Research Study Club of Los Angeles will hold its twenty-seventh Annual Mid-Winter Convention in Ophthalmology and Otolaryngology at the Ambassador Hotel in Los Angeles, January 20-24, 1958.

Frank B. Walsh, M.D., of Baltimore, William F. Hughes, M.D., of Chicago and C. Allen Dickey, M.D., of San Francisco will give lectures in ophthalmology. William J. Endres, M.D., S. Rodman Ir-

vine, M.D., and Russel L. Stimson, all of Los Angeles, and Dr. Dickey will conduct instruction courses.

Otolaryngology lectures will be given by Terrence E. Cawthorne, Esq., F.R.C.S., of England, Francis E. LeJeune, M.D., of New Orleans, Irving B. Goldman, M.D., of New York, and H. Russell Fisher, M.D., Victor Goodhill, M.D., Howard P. House, M.D., Max Pohlman, M.D., Joel J. Pressman, M.D., and Benjamin Simkin, M.D., all of Los Angeles.

E. W. Johnson, Ph.D., of Los Angeles, and Drs. Cawthorne, Goldman and LeJeune will conduct instruction courses in otolaryngology.

Convention registration will begin Sunday, January 19, at 2 p.m. in the Venetian Room at the south end of the main hotel lobby.

FLORIDA

The Fourth Postgraduate Course in Esophageal Speech and Organic Voice Problems, University of Miami School of Medicine, June 16-27, 1958, under the direction of Nathaniel M. Levin, M.D., F.A.C.S., and faculty. Specialists in otolaryngology, physical medicine and rehabilitation and speech therapists are eligible to apply. Government traineeships valued at $250.00 each (tuition and traveling expense) are available on application. For further information, write to Dr. Homer F. Marsh, Dean, University of Miami School of Medicine, Coral Gables, Florida.

IOWA

Dr. Paul Boeder has been appointed to the American Optical Company Chair, in the Department of Ophthalmology of the College of Medicine, State University of Iowa. In his new position as Associate Professor in Physiological Optics, Dr. Boeder will teach and do research at the University.

Dr. Boeder has been associated with American Optical Company since 1935, and has been director of its Bureau of Visual Science since 1941. He is now serving his seventh term as president of the Retina Foundation of Boston, and he is an Honorary Fellow of the American Academy of Ophthalmology and Otolaryngology.

In addition, Dr. Boeder is a member of the American Association for the Advancement of Science, the Optical Society of America, the American Academy of Optometry, and other scientific societies. He has been an instructor and lecturer on ophthalmology at Harvard Medical School since 1950.

Dr. Boeder was born in Hamburg, Germany, in 1902. He received his Master of Arts degree at the University of Pennsylvania in 1925, his Ph.D. at Gottingen in 1931. Prior to his joining the American Optical Company, he was an instructor of mathematics at the University of Delaware from 1926 to 1927 and professor of mathematics at Susquehanna University from 1932 to 1935.

LOUISIANA

A symposium on Uveitis will be the program for the Mid-Winter Convention of the New Orleans Academy of Ophthalmology, February 24 through 28, 1958.

Helenor Campbell Wilder Forester, Michael J. Hogan, M.D., and Samuel J. Kimura, M.D., all of San Francisco, Irving Leopold, M.D., of Philadelphia, Puig Salanas, M.D., of Mexico City, Floyd R. Skelton, M.D., of New Orleans, and Alan C. Woods, M.D., of Baltimore compose the panel.

The registration fee of $75.00 includes associate membership in the New Orleans Academy for the year 1958 as well as all features of the convention. Write to the Executive Secretary, P.O. Box 469, New Orleans, La., for hotel reservations or further information.

NEW YORK

Arthur J. Bedell, M.D., of Albany, N. Y., has just returned from Australia, Tasmania and New Zealand where he was elected an honorary member of the Australian Ophthalmological Society and the New Zealand Ophthalmological Society.

Dr. Bedell spoke in Sydney and Hobart and gave a series of lectures in Melbourne at the meeting of the Australian Ophthalmological Society. During the sessions of the New Zealand Ophthalmological Society in Hamilton, he gave illustrated lectures on ophthalmoscopic subjects.

Bernard Becker, M.D., will give the annual Schoenberg Memorial Lecture before the New York Society for Clinical Ophthalmology and the National Society for the Prevention of Blindness at the New York Academy of Medicine, 8:15 p.m., December 2, 1957.

Dr. Becker, chief of the Department of Ophthalmology at Washington University School of Medicine, St. Louis, will discuss "Tonography: Experimental and Clinical Aspects."

The lecture commemorates Mark J. Schoenberg, M.D., a Rumanian refugee who became a distinguished ophthalmologist. He was a founder of the New York Society and long spearheaded the National Society's glaucoma control program. The two societies have joined for this lecture each year since his death in 1945.

All physicians are welcome.

OHIO

Dr. Herbert J. Rinkel of Kansas City, Missouri, will conduct a three day course on Inhalant and Food Allergy in Akron, Ohio, on December 6, 7, 8, 1957. Other lecturers will be Dr. D. E. Cowen of Denver and Dr. Dor Brown of Fredricksburg, Texas. This course is being sponsored by the Akron Academy of Ophthalmology and Otolaryngology. For further information and reservations contact Dr. Richard H. Stahl, 2674 North Haven Boulevard, Cuyahoga Falls, Ohio.

UNITED KINGDOM

November 30, 1957 is the last day for members of the OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM to plan to read a short paper, give a short pictorial demonstration or show a film before the Annual Congress when it convenes in Cardiff, Wales, April 17, 18 and 19. They should send the title, and an abstract, to the secretary, Mr. K. C. Wybar, 45 Lincoln's Inn Fields, London W.C.2, England, before that date.

The main topic of the meeting, "Complications and Treatment of the Hypermature Cataract," will be discussed initially by Mr. G. T. Willoughby Cashell, Mr. C. A. Cook and Mr. J. R. Hudson. Members who wish to take part in the subsequent discussion are requested to contact Mr. Wybar before the opening of the Congress. Ten minutes is the limit for these talks.

A Symposium on "The Role of the Toxoplasma in Ocular Disease" will feature Professor C. P. Beattie and Dr. J. K. A. Beverley.

The Annual Dinner will be held at the Park Hotel on Thursday, April 17. Members may bring guests.

AMERICAN BOARD OF OPTICIANRY

Theodore E. Obrig and Kenneth N. Ogle, Ph.D., were awarded Beverly Myers Nelson Achievement Awards in Chicago, October 14. Mr. Obrig is past president of Obrig Laboratories, Inc. and Theodore E. Obrig, Inc. Dr. Ogle is professor of physiologic optics at the Mayo Foundation of the University of Minnesota and a member of the Section of Biophysics and Biophysical Research at the Mayo Clinic in Rochester, Minn.

The awards were presented by Leslie W. Myers, Minneapolis, who established the Achievement Awards in Ophthalmic Optics in 1951 in memory of his daughter and as a part of the Educational Foundation in Ophthalmic Optics of the American Board of Opticianry. Two awards are granted each year.

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OPHTHALMOLOGY: One three-year approved residency available in ophthalmology beginning July 1, 1958. Extensive clinical training includes attendance at the Lancaster Course between the junior and senior years. For details apply to Administrator, Baltimore Eye, Ear and Throat Hospital, 1214 Eutaw Place, Baltimore 17, Maryland.

OTOLARYNGOLOGY: One three-year approved residency available in otolaryngology beginning July 1, 1958. Clinical training includes attendance at organized courses in anatomy, endoscopy and endaural surgery. For details apply to Administrator, Baltimore Eye, Ear and Throat Hospital, 1214 Eutaw Place, Baltimore 17, Maryland.

OPHTHALMOLOGIST: Board certified or eligible for Board examinations to take over practice of the late Carl F. Breisacher, M.D. Practice is eleven years old. Modern equipment in downtown office. Write to Mrs. Carl F. Breisacher, no. 14 Druid Place, Charleston 4, West Virginia.

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